THE ROYAL SOCIETY

The health hazards of depleted uranium munitions
Part II

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Preparation of this report

This report has been endorsed by the Council of the Royal Society. It has been prepared by the Royal Society working group on the health hazards of depleted uranium munitions.

The members of the working group were:

Dr Michael R Bailey	Head, Dose Assessments Department, National Radiological Protection Board
Professor Valerie Beral	Professor of Epidemiology and ICRF Cancer Epidemiology Unit, University of Oxford
Professor Dame Barbara Clayton, DBE	Honorary Research Professor in Metabolism, The Medical School, University of Southampton
Professor Sarah C Darby	Professor of Medical Statistics and ICRF Principal Scientist, Clinical Trial Services Unit and Epidemiological Studies Unit, University of Oxford
Professor Dudley T Goodhead	Director, Medical Research Council Radiation and Genome Stability Unit, Harwell
Professor Jolyon Hendry	Head, Experimental Radiation Oncology Group, Paterson Institute for Cancer Research, Christie Hospital, Manchester
Dr Clive Marsh, CBE	Chief Scientist, AWE Aldermaston
Dr Virginia Murray	Director, Chemical Incident Response Service, Guy's and St Thomas' Hospital NHS Trust
Professor Barry Smith	British Geological Survey
Professor Brian Spratt FRS (Chair)	Wellcome Trust Principal Research Fellow, Department of Infectious Disease Epidemiology, Imperial College School of Medicine
Professor Marshall Stoneham FRS	Massey Professor of Physics, Department of Physics and Astronomy, University College London

Secretariat

Ms Sara Al-Bader, Dr Peter Collins, Dr Nick Green, Dr Mark Wilkins (Science Advice Section, Royal Society)

Preface

Following the large-scale deployment of depleted uranium (DU) munitions in the Persian Gulf and reports that these weapons were used in Kosovo (subsequently confirmed), the Royal Society set up a working group to provide an independent scientific assessment of the health hazards of DU munitions. The working group has produced the first part of its report, which considers the radiological consequences of exposure to DU (Royal Society 2001). This is the second part of the report. It considers other possible health consequences of the use of DU munitions and their impact on the environment. Several other independent reports have recently considered these issues (eg UNEP 1999, 2001; Fulco et al 2000; WHO 2001).

The first authenticated use of DU munitions during a military conflict was in the Gulf War. Soon after this conflict there were reports of illness in soldiers who served in the Gulf War, typically involving pain, fatigue, irritability and sleep disturbances; this became known as Gulf War Syndrome. Increased illness among soldiers following military campaigns has previously been documented, but illness following the Gulf War appears to be particularly common. In a recent survey about 17% of UK soldiers who served in the Persian Gulf considered that they have Gulf War Syndrome (Chalder et al 2001). Apart from the trauma of war, soldiers in the Gulf were subjected to a number of potentially toxic exposures, including multiple vaccinations, squalene, antidotes to chemical warfare agents, insecticides and rodenticides, smoke from burning oil wells, solvents and lubricants, as well as to aerosols containing DU arising from the use of DU munitions (Unwin et al 1999; Fulco et al 2000; Hotopf et al 2000; Cherry et al 2001a,b; Kang and Bullman 2001; Reid et al 2001). It has been difficult to associate Gulf War Syndrome with any of the above potential exposures, although associations between disease and the number of vaccinations, squalene and the use of antidotes to chemical warfare agents have been suggested (Cherry et al 2001b; Reid et al 2001). To date, the published studies on the health of veterans have not considered exposure to DU to be a major contributor to Gulf War Syndrome. However, DU is radioactive and toxic and if exposures are sufficiently high it could increase the incidence of cancer, damage the kidneys or have other adverse health effects.

In this second part of the report we focus on the possible effects of the use of DU munitions on the kidney, as uranium is a nephrotoxin and the kidney will be the organ most at risk from exposure to high levels of DU on the battlefield. We also consider whether the use of large amounts of DU in military conflicts (at least 300 tons in the Gulf War, CHPPM 2000) will have long-term effects on the environment that constitute a continuing health hazard for those who live in, or return to, areas where DU munitions were deployed.

In June 2001 an open public meeting was held to consider Part I of the Royal Society report. In this part of the report (Part II) we respond to some of the concerns that were raised at this meeting, or in correspondence or discussions with members of the working group. Part I of the report considered only the radiological risks of cancer arising from exposure to DU and there were concerns that radiation may also have adverse effects on the immune system or on reproductive health. Part II of the report therefore considers these latter issues, although its main focus is on the adverse effects that may arise from the chemical toxicity of uranium. It has been suggested by some veterans that intakes of DU in the Gulf War for some soldiers involved in inspecting and salvaging vehicles struck by DU munitions may have been even greater than we considered in Part I. We consider intakes for these soldiers and also evidence provided on uranium isotope measurements and adverse health effects.

Summary

There has been much concern about the health consequences of the use of depleted uranium (DU) munitions during military conflicts in the Persian Gulf and the Balkans, and of the longer term effects for those living in areas where DU munitions are deployed. The Royal Society therefore convened an independent expert working group to review the present state of scientific knowledge about the health and environmental consequences of the use of DU munitions, in order to inform public debate.

The first part of the report was published in May 2001 and covered the radiological consequences of exposures to DU on the battlefield. This is Part II of the report, which considers adverse health effects from the chemical toxicity of uranium, the non-malignant radiological effects of DU intakes and the impact on the environment. After publication of Part I there was a public meeting to discuss the report, and at this meeting, and in further consultations and correspondence with scientific experts and veterans, a number of issues were raised which we examine here.

Chapter 1 considers the possible adverse effects of DU exposure that arise from the chemical toxicity of uranium. Full details are given in Appendix 1. It is well established from animal studies, and from human exposures, that the kidney is the organ most susceptible to the toxic effects of uranium. A large body of literature exists about the toxic effects of inhaled, ingested and injected uranium compounds on laboratory animals. However, there are large differences in the susceptibilities of animal species to uranium, which make it difficult to use the animal data to estimate the intakes of uranium that have adverse effects in humans.

There are few studies of humans exposed to substantial intakes of uranium and hence the concentrations of uranium in the kidney that lead to serious adverse effects are not well documented. Very few humans have had sufficiently large acute intakes of uranium compounds to lead to kidney failure. Studies of these few cases indicate that kidney failure is likely to occur within a few days at concentrations above about 50 micrograms uranium per gram kidney.

The chronic levels of kidney uranium that lead to minor kidney dysfunction in humans (measurable by sensitive biochemical tests of kidney function) are not well established, but are considered to be at least ten-fold less than the value of three micrograms uranium per gram kidney that has often been used as the basis for occupational exposure limits. Acute exposures that lead to concentrations of about 1 microgram uranium per gram kidney have been associated with minor kidney dysfunction, but the levels of kidney uranium that can occur for a short period without causing long-term adverse effects on the kidney have not been defined.

The available evidence suggests that there is little, if any, increase in kidney disease among workers involved in the processing of uranium ores or in uranium fabrication plants. However, this is not necessarily reassuring, since the daily intakes that occur from chronic inhalation exposure to uranium particles in these industries would typically have been much lower than the acute intakes that might be received by the most heavily exposed soldiers in a military conflict. Also, the typical forms of the inhaled particles in industrial settings and on the battlefield will be different, and these alternative forms might not have the same adverse effects.

There are no data on the long-term effects of the use of DU munitions on humans and the environment because they were first used in a military conflict in 1991 during the Persian Gulf War. Consequently, the long-term risks to health and the environment have been evaluated in the absence of data over appropriate timescales.

We have drawn the following conclusions about the risks from the chemical toxicity of uranium:

- The estimated DU intakes for most soldiers on the battlefield are not expected to result in concentrations of DU in the kidney that exceed 0.1 microgram per gram kidney, even transiently. Consequently, in these cases it is not expected that adverse effects on the kidney or any other organ would occur.
- Levels of uranium in the kidneys of soldiers surviving in tanks struck by DU rounds, or of soldiers working for protracted periods in struck tanks, could reach concentrations that lead to some short-term kidney dysfunction, but whether this would lead to any long-term adverse effects is unclear as adequate studies of the long-term effects on the kidney of acute exposures to elevated levels of uranium are not available. According to worst-case assumptions, kidney uranium levels in some of these soldiers could be very high, and would probably lead to kidney failure within a few days of exposure. We are not aware of any cases of kidney failure, occurring within a few days of exposure, in US soldiers who would have received the highest DU intakes during the Gulf War, but we cannot rule out significant kidney damage for a few soldiers under worst-case assumptions.
- The kidney is a resilient organ and about two-thirds of kidney function can be impaired without obvious clinical signs of disease. Similarly, apparently normal kidney function can be restored even after a large

acute intake of uranium. This raises difficulties when assessing the health of Gulf War veterans, since large intakes of DU, which could increase the chance of lung cancer or kidney disease in later life, would probably not be apparent from a clinical examination or from standard blood and urine analyses carried out several years after exposure. For those who may have been exposed at some time in the past to substantial intakes of DU, an analysis of uranium isotopes is required to assess intakes and any possible health consequences.

- Large inhalation intakes of DU particles may result in short-term respiratory effects, as would a large intake of any dust, but long-term respiratory effects are not expected, except perhaps for the most heavily exposed soldiers, under worst-case assumptions, where some fibrosis of the lung may occur from radiation effects, in addition to an increased risk of lung cancer that was discussed in Part I of the report.
- Uranium is deposited in bone but there is insufficient evidence to conclude whether large intakes of DU on the battlefield could have adverse effects on the bone.
- Although there is no clear evidence that occupational exposures to uranium have consequences for reproductive health, effects on reproductive health have been observed in mice after high intakes of uranium. Accordingly, epidemiological studies of the reproductive health of Gulf War veterans and of the Iraqi population are underway. If effects are seen then further investigation would be required to determine the relative contributions from DU and from other possible causes.

Chapter 2 considers the environmental effects of the use of DU munitions. Full details are given in Appendix 2. After a conflict in which large amounts of DU munitions are deployed, those who return to live in the area will be exposed to both resuspended DU particles and to contaminated food and water supplies.

We have therefore assessed the long-term effects on the environment.

- Contamination will occur mainly from DU particles and penetrator fragments deposited in the soil, and from intact penetrators buried in the ground. The movement of DU from these sources into susceptible components of the environment will depend on a number of factors, including the rates of corrosion, which depend on soil properties, the amount of resuspension of soils, and the proximity of DU penetrators to surface soils and water sources that feed into local water supplies. These sorts of factors will also influence the extent of uptake of DU by plants and intakes by local food animals.
- The levels of environmental contamination will be very variable, which makes it difficult to generalise about levels of DU intakes. These levels could range from being so small that they do not materially increase the concentration of uranium naturally present in the environment to worst-case scenarios, such as a penetrator lodging directly in contact with groundwater, which could feed uranium directly into a local water supply, such as a well.
- Initially, exposure of the local population will be to DU particles resuspended from contaminated soil, and from contaminated water and food, but the inhalation exposure and intakes from food will decrease, and the proportion of exposure from intakes of DU from contaminated water sources will increase.
- Measurements of environmental contamination in Kosovo have not shown widespread contamination with DU, although hot spots of contamination are present around penetrator impacts. However, most of the DU deployed in a military conflict remains in the ground and environmental movement of DU from buried penetrators will be slow. Long-term monitoring of uranium contamination in water supplies therefore needs to be carried out in areas where DU munitions were deployed.
- We have estimated the intakes by inhalation of resuspended DU particles for both children and adults. For those returning to live in areas where DU munitions were deployed, the inhalation intakes from resuspended DU are unlikely to cause any substantial increase in lung cancer or any other cancers. The estimated excess lifetime risk of fatal lung cancer is about one in a million, although there would be higher risks for some individuals with worst-case intakes of DU due to higher levels of local contamination. Estimated risks of other cancers are at least 100-fold lower.
- Similarly, no effects on kidney function are expected for most individuals, although small effects on kidney function are possible using worst-case assumptions, but would at most only apply to a small number of individuals.
- Ingestion of DU in contaminated water and food, and from soil, will be highly variable but may be significant in some cases, eg children playing in areas where a DU penetrator has impacted or where a penetrator feeds uranium into a local water supply.

Chapter 3 considers some of the issues that were raised at the public meeting following the publication of Part I of the report. We also consider further evidence provided to the working group on levels of exposure to DU, uranium isotope measurements and health problems of Gulf War veterans.

One issue raised at the public meeting was the possibility of effects on the immune system from inhaling DU particles. Effects on components of the immune system have been observed in humans and animals exposed to large intakes of radioisotopes that

irradiate the red bone marrow. The levels of irradiation of the red bone marrow for all DU exposure scenarios are predicted to be less than those from background sources, except for Level I and II worst-case scenarios, where they could be considerably higher than background levels, but would still be too low to cause effects on the immune system that would increase susceptibility to infection.

Evidence was taken from Dr Doug Rokke who was part of a unit involved in assessing battlefield damage and in cleaning up struck allied and Iraqi tanks after the Gulf War. Dr Rokke considers that for a number of reasons the intakes for soldiers involved in these activities would have been substantially higher than we proposed. Some of these claims conflict with those in military reports. However, we have provided estimates of DU intakes, and of the risks of cancer and adverse kidney effects, for these proposed levels of exposure.

If these very large exposures to DU are realistic, a small number of soldiers who worked for very long periods cleaning up vehicles struck by DU munitions during the Gulf War might have suffered adverse health effects, involving kidney damage and a substantial increase in the risk of lung cancer.

Measurements of uranium isotopes in the urine of some veterans have been carried out by Dr Pat Horan in Canada. These results were presented to the working group by Dr Asaf Durakovic and in discussions it became clear that there are uncertainties about the reliability of these measurements of DU in urine, due to the absence of an appropriate control group and the difficulties associated with obtaining isotope ratios from samples of urine containing small amounts of uranium.

Reliable measurements of DU in urine are important as even ten years after the Gulf War they probably could still provide an assessment of intakes and risks.

Recommendations

- The need for further information about the intakes of DU that occur on the battlefield and the properties of DU aerosols was highlighted in Part I of the report. This information is also required to assess the levels of uranium in the kidney and to predict more precisely the likely effects on health of the chemical toxicity of uranium.
- We have previously recommended long-term epidemiological studies of soldiers exposed to DU aerosols, or with retained DU shrapnel, to detect any increased incidence of cancers. These long-term studies are also required to detect any increased incidence of non-malignant lung disease and kidney disease in later life.
- Any studies of individuals who might have received substantial intakes of DU must include the most sensitive modern biochemical methods to detect signs of kidney dysfunction and should involve an expert nephrologist.
- A small number of veterans in the Gulf War working for protracted periods in struck vehicles could have received large intakes of DU. There are anecdotal reports of deaths and illness in these veterans and an independent study of mortality and morbidity among these veterans is required.
- There are reports that DU has been detected in the urine of some Gulf War veterans but the reliability of the available measurements is subject to considerable uncertainty. A carefully validated method for measuring uranium isotope ratios in urine containing small amounts of uranium is required. These studies should be conducted at independent laboratories with the collaboration of veterans' groups. Such studies are being progressed by the MOD's DU Oversight Board.
- In any future conflict using DU munitions, measurements of urinary uranium and sensitive modern biochemical tests of kidney function need to be carried out as soon as possible after exposure on soldiers who are exposed to substantial intakes of DU.
- Serious effects on the kidney and lung are possible under worst-case assumptions for a few soldiers who could receive large acute exposures to DU on the battlefield. Any case of acute kidney failure occurring within a few weeks of exposure should be thoroughly investigated to establish whether high kidney uranium levels could be the cause.
- Areas should be cleared of visible penetrators and DU contamination removed from areas around known penetrator impacts.
- Long-term environmental sampling, particularly of water and milk, is required and provides a cost-effective method of monitoring sensitive components of the environment, and of providing information about uranium levels to concerned local populations. Monitoring may need to be enhanced in some areas, by site-specific risk assessment, if the situation warrants further consideration.
- The environmental behaviour of the corrosion products of DU-titanium alloys and particles should be compared with that of naturally occurring uranium minerals.
- Information should be obtained on the bioavailability of DU-Ti products from DU munitions and their corrosion products (particles, metallic fragments and secondary precipitates associated with the corrosion process), and on whether bioconcentration of these materials occurs in local food animals or plants.

1 Non-radiological health effects from exposure to DU munitions

1.1 Introduction

The general properties of uranium and DU, and the use of DU rods as kinetic energy penetrators in munitions designed to pierce the heavy armour of modern battle tanks, have been described in Part I. The deployment of DU munitions on the battlefield can result in exposure of soldiers or local inhabitants to DU by a number of routes. For soldiers, the most important of these is the inhalation of DU particles in aerosols produced when DU penetrators pierce hard targets, and the presence of retained DU shrapnel, although ingestion of DU may also be an important exposure route. Inhalation results in the deposition of small particles of oxidised DU in the lung and the translocation of some of these particles to the associated lymph nodes. The radiation emitted from these particles might increase the probability of lung cancer, and cancers of some other tissues or organs, and the extent of the increased lifetime risks of various cancers for different intakes of DU has been considered in Part I. Internalisation of DU will also result in increased levels of uranium[1] in body tissues, which might have adverse effects arising from its chemical toxicity. These effects are likely to be mainly on the kidney as this is believed to be the organ most at risk from elevated levels of uranium. We also consider other non-malignant adverse effects that might be caused by exposure to DU.

Uranium occurs naturally in the environment. The concentrations of uranium in water, food and soils vary considerably, but are typically 0.1-5 µg per litre, 0.01-2 µg per kg and 0.1 µg - 2 mg per kg, respectively[2]. Uranium particles are also present at low concentration in air (0.01-3 ng per cubic metre of air), mainly from resuspension of soil. Typical natural intakes of uranium are about 1 µg per day and the majority of this is from food and water. However, in most countries the range of intakes varies by a factor of about ten. Much greater intakes of natural uranium occur in some regions, due to high uranium content in local rocks, proximity to uranium mining or the use of drinking water from private sources that contain high levels of uranium.

In military conflicts involving DU munitions the main concern is from the inhalation of DU particles in aerosols arising from impacts of DU penetrators with their targets. As discussed in Part I, there are considerable uncertainties about the amounts of DU that may be inhaled, the fraction that may gain access to the lungs, and the rates of dissolution of those particles of DU that may be retained in the lung or translocated to the associated lymph nodes. The rate of dissolution of DU particles is an important parameter as the radiation received by the lungs and associated lymph nodes from an intake of DU will be highest if the inhaled DU particles are highly insoluble. In contrast, for the toxic effects, the highest levels of DU in the kidney will occur if the inhaled particles are highly soluble.

The main forms of uranium released during impacts of DU munitions with their targets have been reported to be triuranium octaoxide (U_3O_8), uranium dioxide (UO_2) and possibly amorphous uranium oxide. Combustion of DU results almost entirely in the formation of U_3O_8. As discussed in Part I of this report, a proportion of the DU retained in the lungs and lymph nodes is believed to dissolve relatively quickly whereas the majority dissolves very slowly. There is, however, considerable uncertainty about the fraction of DU in aerosols released from impacts and fires that dissolves rapidly in body tissues.

The uncertainties in the amounts of DU that may be inhaled, the size distribution of DU particles within the aerosols and the proportion of the retained DU that dissolves rapidly result in a wide range of possible levels of uranium that could occur in the kidney. Our approach has been to use the central estimates of intakes from information in the published reports, and the central estimates of the other parameters that affect the amount of DU reaching the kidney, for a limited number of possible battlefield scenarios. Biokinetic models can then be used to calculate the levels of uranium in the kidney at any time after the intake to provide a central estimate of the kidney uranium concentrations. These models have been developed and refined using a large body of data from animal studies, and from human volunteer studies, and provide the only well-validated way of relating intakes of uranium to the levels that will occur in organs and tissues of the body (Part I, Annexe A). A further discussion of the utility of the modelling approach to assessing risks is given in Chapter 3.

We also use intakes of DU that we consider are unlikely to be exceeded, and the values of the other parameters that maximise the levels of uranium reaching the kidney, to provide a 'worst-case' estimate of kidney uranium concentrations.

[1]DU and natural uranium are not distinguished as they differ only in isotopic content, which does not affect their chemical properties or their toxic effects on the kidney or other organs.

[2] ng, nanogram (one thousand millionth part of a gram); µg, microgram (one millionth part of a gram); mg, milligram (one thousandth part of a gram); kg, kilogram (one thousand grams).

The predicted maximum levels of uranium in the kidney for different battlefield scenarios were estimated in Part I (Appendix 1, table 27). For these levels of uranium in the kidney, it should be possible to estimate the likely effects on kidney function. In practice this is problematic, as there is very little information that relates levels of uranium in the human kidney to clinical symptoms and biochemical indicators of kidney function. Direct measurement of uranium concentrations in the human kidney, or microscopic examination of kidney tissue, by obtaining a sample of the kidney (biopsy) might be harmful and therefore is not advisable. There is a very extensive literature on the effects of uranium on experimental animals but this has to be treated with considerable caution as the levels that result in kidney (or other) damage in humans may be different from those in laboratory animals.

Additionally, most (if not all) studies on the human toxicity of uranium relate to the effects on adults. In some military conflicts where DU is deployed, and in the aftermath of conflicts, there could be exposure of mothers and foetuses, infants and children to elevated levels of uranium. Animal studies suggest that absorption of uranium from the gut of neonates might be higher than in older children or adults (ICRP-69 1995). Furthermore, there are studies indicating increased absorption of uranium from the gut of fasted animals (ICRP-69 1995), which raises the possibility that levels of uranium in the kidney may reach higher levels in individuals who are malnourished as a consequence of war.

1.2 Toxicological effects of uranium

The kidney is considered to be the main target organ for the chemical toxicity of uranium. Uranium accumulates in the renal tubular epithelium and causes cellular necrosis and atrophy in the tubular wall leading to decreased reabsorption of amino acids and small proteins by the renal tubules (reviewed in Leggett 1989).

Many studies on the toxicity of uranium in laboratory animals have been carried out since the 1940s. These provide a wealth of information on the intakes of soluble and insoluble uranium compounds that produce adverse effects in a range of laboratory animals, by ingestion, inhalation, injection and by application to the skin. In general, much lower amounts of a uranium compound are required to produce toxic or lethal effects by intravenous injection than by ingestion or inhalation, since all of the injected uranium directly enters the bloodstream, whereas only a fraction of the ingested or inhaled uranium enters the bloodstream and reaches the kidney. For similar reasons, highly soluble uranium compounds are more toxic than compounds with low solubility.

Substantial differences occur between the concentrations that produce toxic effects in different animals, which makes the extrapolation of animal results to humans subject to considerable uncertainties. Estimates of the lowest uranium concentrations that alter kidney morphology or kidney function have been reported to be as high as 1 µg uranium per gram kidney in the rat (Diamond et al 1989), about 0.3 µg per gram in the dog (Morrow et al 1982) and as low as 0.02 µg per gram in the rabbit (Gilman et al 1998a). Even studies carried out by the same research group, using the same experimental protocols, have lead to very different results for different animal species and substantial differences for the same species. For example, in the recent studies of Gilman et al (1998a,b), the lowest observed adverse effect on the kidney in pathogen-free male New Zealand white rabbits occurred at chronic intakes of about 1.4 mg uranium per kg per day, whereas adverse effects were observed at intakes of about 0.05 mg per kg per day in similar rabbits that were not selected as being pathogen-free. Males and females can also differ in their susceptibility to uranium. Gilman et al (1998a) found that female New Zealand white rabbits were five times less susceptible to chronic exposures to soluble uranium than similar male rabbits. The reasons for these large variations in susceptibility to the nephrotoxic effects of chronic ingestion of soluble uranium are not understood, but the studies highlight the difficulties in precisely defining the lowest uranium intake that results in an adverse effect on the kidney even for a single strain of a single species.

In contrast with the extensive literature on the effects of uranium on animals there are very few detailed studies of the effects of substantial intakes of uranium on humans. These studies are reviewed in Appendix 1. The human studies that provide the basis of our knowledge of the toxicity of uranium differ from the animals studies in the way that adverse effects are defined. In animals, the lowest concentrations that have adverse effects are typically defined by morphological examination of kidney tissue, which is not feasible for studies of humans exposed to elevated levels of uranium, where biochemical tests of kidney function are used. The relative sensitivities of these two approaches are not clearly documented.

Most of the reports of human exposures to uranium that do exist in the published literature describe acute exposures to large intakes during accidents in the uranium industry, but some describe controlled intakes by volunteers. There are also studies of the consequences of chronic exposure to lower concentrations of uranium. In addition, there are a number of large-scale epidemiological studies of deaths from kidney disease among workers in the uranium industry where elevated exposure to uranium will have occurred.

1.3 Exposure limits

1.3.1 Exposure limits for the public: ingestion (WHO 2001)

Experimental studies with rabbits and rats, particularly those of Gilman et al (1998a,b,c), have identified daily intakes of soluble ingested uranium compounds where effects on the kidney become apparent over a 91-day period. Recommended safety limits for the ingestion of uranium by humans have been obtained by WHO (and others) by using the daily intakes from these animal experiments that produce no apparent effect on the kidney, or are the lowest daily intakes that produce an observable effect on the kidney (WHO 2001). These levels are reduced by an uncertainty factor that, among other things, takes into account possible differences in the susceptibility of laboratory animals and humans to the toxic effects of uranium, differences in the amounts of uranium reaching the kidney and limitations in the key animal studies.

The lowest daily intake of soluble uranium that results in observable effects on the rat or rabbit kidney is about 50 µg per kg body mass per day. This value is reduced by a factor of 100 (the default uncertainty factor) to provide the WHO safety limit (the tolerable daily intake) for the chronic ingestion of soluble uranium for humans (0.5 µg per kg body mass per day – about 35 µg per day for a 70 kg (11 stone) human).

Ingestion of insoluble uranium compounds is less toxic as a smaller proportion of the intake accumulates in the kidney, and the proposed WHO safety limit is 5 µg uranium per kg body mass per day (350 µg per day for a 70 kg human).

1.3.2 Exposure limits for the public: inhalation (WHO 2001)

The toxicity of inhaled uranium compounds is dependent both on the particle size and on the solubility of the uranium compound. To gain access to the lung, particles need to be in the respirable range (less than a few micrometres in diameter); most larger particles deposit in the upper airways and are removed by normal mucociliary flow and swallowed. Inhaled particles of highly insoluble uranium compounds will be very slowly absorbed into the blood whereas inhaled particles of soluble uranium compounds will be rapidly absorbed into the blood. Thus, following inhalation of the same mass of uranium, there will be a higher concentration of uranium in the kidney for the soluble compound than the insoluble compound. For some compounds of uranium, and for the mixtures of compounds that might arise in impacts or fires involving DU munitions, a fraction of the material will be absorbed into the blood rapidly, and the rest much more slowly.

A large number of animal studies have been carried out on the effects of inhalation of soluble and insoluble uranium compounds. These suggest that chronic inhalation of air containing about 0.2 mg uranium per cubic metre may result in slight damage to the kidney. Application of a number of corrections (differences in breathing rates, etc), and an uncertainty factor of 100, results in a tolerable daily intake for the inhalation of soluble and moderately soluble uranium compounds of 0.5 µg per kg body mass per day (about 35 µg per day for a 70 kg human).

The inhalation of 5 mg per cubic metre of insoluble uranium compounds (UO_2) by dogs and monkeys for several years resulted in no observable effects on the kidney (Leach et al 1973), and a tolerable daily intake for man of 5 µg insoluble uranium per kg body mass per day has been proposed (350 µg per day for a 70 kg human). This limit is appropriate for chemical toxicity but it would result in a total radiation dose above the radiation exposure limit for the general public (one millisievert per year), and it has been suggested (WHO 2001) that the inhalation limit for insoluble uranium compounds should be the same as that for soluble compounds (0.5 µg per kg body mass per day).

These tolerable daily intakes for the general public correspond to the inhalation of about 1 µg of uranium particles in the respirable range per cubic metre of air. The suggested occupational limit for inhalation of soluble or insoluble uranium compounds is about 50 times greater than that for the general public (WHO 2001).

1.3.3 Occupational exposure limits

Occupational toxicological exposure limits based on 3 µg of uranium per gram kidney have often been cited but appear to have been derived primarily from radiological considerations, rather than any solid body of evidence that indicates an absence of any toxic effects on the human kidney, or any other organ or tissue, below this level. In several studies with laboratory animals kidney damage is apparent following uranium intakes that result in less than 3 µg of uranium per gram kidney (Diamond et al 1989; Leggett 1989; Gilman et al 1998a,b,c). The limited human data (see below) also indicate that biochemical indicators of kidney dysfunction may be elevated at levels below 3 µg of uranium per gram kidney.

Occupational limits for long-term exposure published by various regulatory bodies range from 0.05 to 0.2 mg per cubic metre of air for soluble uranium and from 0.2 to 0.25 mg per cubic metre of air for insoluble uranium (Appendix 1, Section 2.4). WHO (2001) has suggested a limit of 0.05 mg per cubic metre of air (eight-hour time-weighted average) for both soluble and insoluble uranium, to take account of both radiation and chemical effects.

1.4 Toxicity of uranium in humans

There are a number of studies that can be used to understand the levels of uranium that are toxic to humans. Some of these are studies of individuals, or groups of individuals, who have been exposed for long periods to elevated levels of uranium in their water supply, or from their occupation (chronic exposures). These exposures are of particular relevance to the health of soldiers with retained DU shrapnel which, by slow dissolution, leads to chronically elevated levels of uranium in the kidney, or to some situations where increased intakes could occur among the local population due to DU contamination of water or food supplies following a conflict. In most cases the exposures on a battlefield will occur over a short period of time (acute exposures) and uranium levels in the kidney will rise to a peak and then decline. There are a number of studies of humans who have received substantial acute exposures to uranium, which are particularly relevant to the health consequences from the typical intakes of DU that occur on the battlefield.

1.4.1 Chronic exposures

1.4.1.1 Drinking water containing high uranium concentrations

Some indication of the lowest kidney uranium concentration that results in nephrotoxicity in humans can be obtained from the studies of Limson Zamora et al (1998). They studied kidney function in a group of individuals chronically exposed to low levels of uranium in drinking water from public supplies (less than 1 µg per litre) or to high levels of uranium from private wells (2-780 µg per litre). Significant differences in the results of some kidney function tests were identified among the heavily exposed group, which correlated with the extent of their uranium intakes.

From these human data it is possible to relate the adverse effects detected by kidney function tests to the estimated levels of uranium in the kidney using the current International Commission on Radiological Protection (ICRP) biokinetic model for uranium (Part I, Annexe A, Section A2.1). Figure 1.1 shows that after one year of constant uptake to blood of 1 µg per day, the level of uranium is predicted to reach 0.0056 µg per gram kidney and after 50 years it would reach 0.011 µg per gram kidney.

For uranium in soluble form it is generally assumed that 2% of the uranium ingested by adults is absorbed into the blood (ICRP-69 1995, Part I, Appendix 1, Annexe A). Thus it is predicted that the kidney uranium levels shown in figure 1.1. would be reached from constant ingestion of 50 µg per day of soluble uranium. These values can be scaled up to estimate the levels of uranium in the kidneys of the individual with the highest average daily intakes of soluble uranium (570 µg of uranium per day) in the study of Limson Zamora et al (1998). After one year of chronic exposure, the level of uranium in this individual is predicted to reach 0.06 µg per gram kidney and after 50 years of daily exposure it would reach 0.13 µg per gram kidney. As subtle effects on the kidney were observed in individuals with lower uranium intakes than this maximally exposed individual, it is likely that slight adverse effects on the kidney would be observed at levels below 0.1 µg uranium per gram kidney.

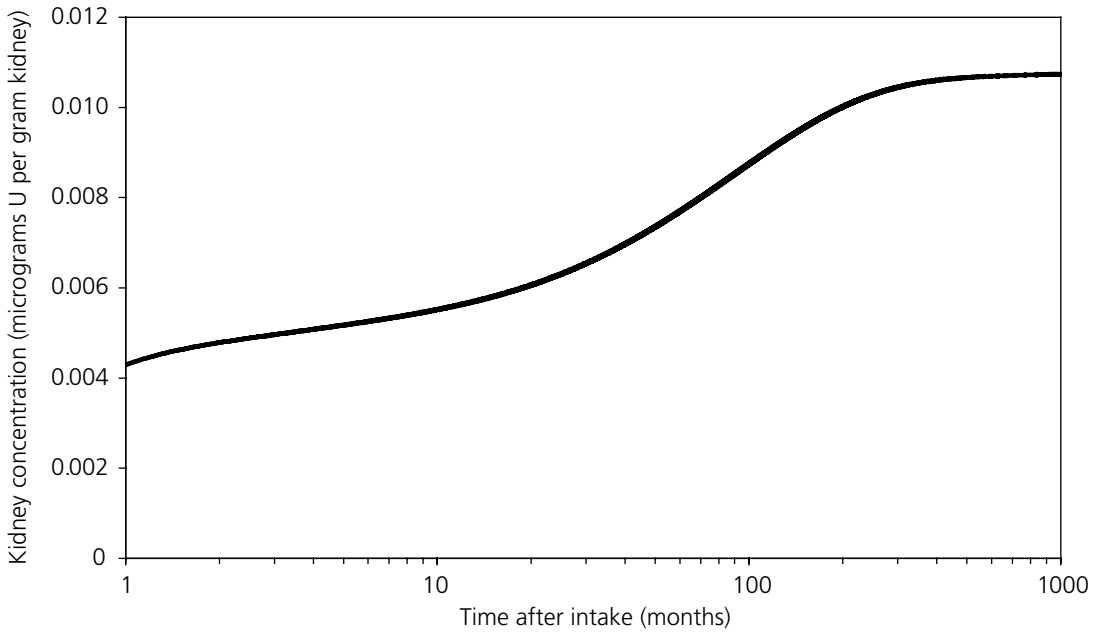
1.4.1.2 Chronic exposure of uranium mill workers

Thun et al (1985) have reported reduced renal proximal tubular reabsorption of amino acids and low molecular weight proteins consistent with uranium nephrotoxicity among a small group of uranium mill workers who had relatively high exposures to soluble uranium. In these workers 21% of their urine samples contained more than 30 µg uranium per litre and some individuals excreted about four times this level. Assuming an output of 1.5 litres of urine per day, the workers exceeding this level of urinary uranium would have at least 0.25 µg uranium per gram kidney (Annexe A, Section A2.2) and the highest level would be about 1 µg per gram. The signs of kidney damage in the workers are therefore consistent with the view that chronic exposures that lead to concentrations less than 3 µg uranium per gram kidney are nephrotoxic. The lack of data on the uranium levels in urine for individual workers in relation to their kidney function tests precludes a more precise assessment of the uranium levels causing toxicity.

1.4.1.3 Soldiers with retained DU shrapnel

The group of US soldiers involved in 'friendly fire' incidents that have retained DU shrapnel provide further information about the chronic effects of uranium in humans. From the data of Hooper et al (1999) and McDiarmid et al (2000), the highest urinary excretion among the veterans with retained DU shrapnel was estimated to be about 60 µg uranium per day (Annexe A, Section A2.3). Most of the uranium entering the blood is excreted in the urine and therefore the rate of uptake of uranium to the blood is approximately equal to the urinary excretion rate. From figure 1.1, an uptake rate of 1 µg uranium per day gives a kidney uranium concentration of 0.0056 µg per gram kidney at one year and 0.0090 µg per gram kidney at ten years. For the soldier with the highest level of uranium entering the blood from DU shrapnel (60 µg per day) we therefore predict about 0.3 µg uranium per gram kidney at one year and about 0.5 µg uranium per gram kidney at ten years. Measurements between 1993 and 1995 (Hooper et al 1999) showed an average urinary excretion rate of about 10 µg per day for the soldiers with retained shrapnel, which would be predicted to result in 0.06 µg uranium per gram kidney at one year and 0.1 µg uranium per gram kidney at ten years.

Figure 1.1. Predicted concentration of uranium in the kidney from the constant uptake into the blood of 1µg uranium per day.



At present there are no published reports of kidney dysfunction in the soldiers with retained DU shrapnel. This is slightly inconsistent with the study of Limson Zamora et al (1998) where some adverse effects were observed at predicted kidney uranium levels about four times lower than the highest kidney concentration predicted for the soldiers with DU shrapnel.

1.4.2 Acute exposures

The ingestion of relatively large amounts of soluble uranium is required to kill laboratory animals. In rats and mice ingestion of 114-136 mg of soluble uranium per kg body mass resulted in the death of 50% of the animals (Domingo et al 1987). Extrapolation to humans is subject to much uncertainty, as discussed above, but this would correspond to ingestion of about 9 g of soluble uranium for a 70 kg man. Insoluble uranium compounds are much less toxic when ingested as smaller amounts of uranium occur in the kidney.

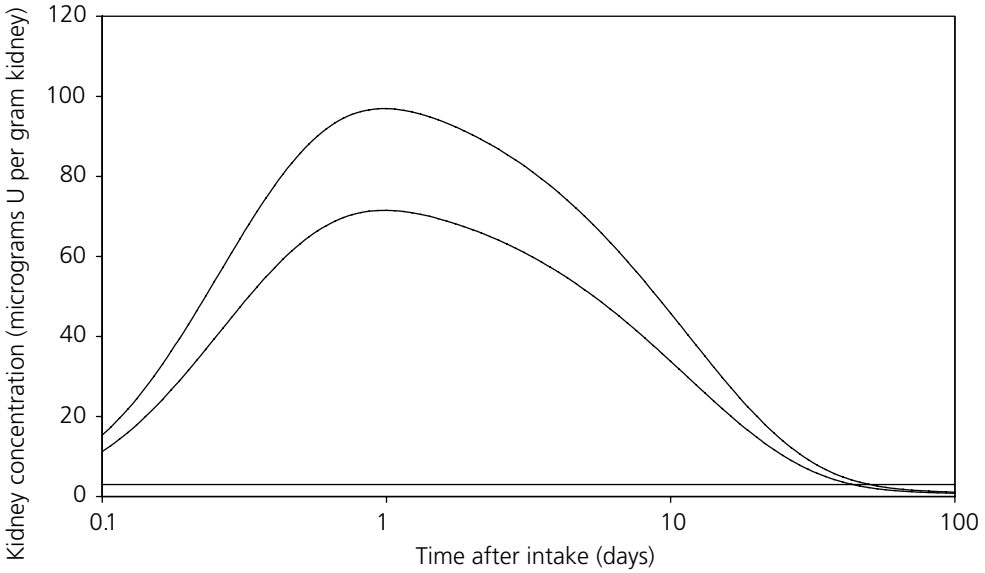
The concentrations of uranium in the human kidney that lead to severe or life-threatening effects on the kidney (and other organs) can be obtained from studies of acute exposures to high levels of uranium. There are few reports where levels of uranium in the kidney at different times after exposure can be estimated and related to clinical symptoms and to biochemical markers of kidney dysfunction. One of the most illustrative studies of the consequences of the ingestion of soluble uranium is provided by an individual who attempted suicide by ingesting about 15 g of uranium acetate (Pavlakis et al 1996). The individual suffered severe kidney dysfunction and required dialysis for two weeks before sufficient kidney function was recovered, and also suffered from anaemia, and effects on the intestines, heart and liver. He remained anaemic for about eight weeks and biochemical signs of kidney dysfunction remained for six months.

Using the current ICRP biokinetic model for uranium it is estimated that an acute intake of 8.5 g of soluble uranium (equivalent to 15 g of uranium acetate) would result in a peak concentration of about 100 µg uranium per gram kidney (figure 1.2). The estimated levels of uranium within the kidney would remain above 3 µg uranium per gram kidney for at least 50 days.

This case report indicates that an acute intake of uranium that is estimated to result in a peak concentration of about 100 µg per gram kidney has very serious effects on kidney function, requiring haemodialysis, and results in prolonged kidney dysfunction.

An accident described by Zhao and Zhao (1990) involved an individual with very extensive skin exposure to a solution of hot uranyl nitrate and uranium dioxide. The level of uranium in urine increased rapidly and the patient became critically ill with severe kidney dysfunction. After one month the patient had recovered and kidney function was normal but he complained of tiredness, dizziness and headaches over the next seven years. This intake of uranium is predicted to have resulted in a maximum concentration of about 35 µg uranium per gram kidney, with the uranium concentration remaining above 3 µg per gram kidney for about 40 days (Annexe A, Section A3.3). The case report suggests that a peak kidney uranium concentration of about 35 µg per gram can cause serious kidney dysfunction, but the extensive burns sustained by this individual would almost certainly have contributed to his critical condition.

Figure 1.2. Predicted uranium concentration in the kidneys following the ingestion of 15 g of uranium acetate. The two curves show the uranium concentration according to two different estimates of the fraction of the uranium absorbed from the gut to the blood (see Annexe A, Section A3.1). A solid horizontal line indicates a kidney uranium concentration of 3 µg per gram as this has been used as the basis for occupational exposure limits.



Zhao and Zhao (1990) described another individual who accidentally inhaled a large amount of uranium tetrafluoride (a moderately soluble uranium compound). Levels of uranium in urine increased over the first two months to reach a maximum of approximately 3 mg of uranium per litre of urine and gradually reduced to reach normal levels three years after the accident. This intake of uranium is predicted to have resulted in a maximum concentration of about 10 µg uranium per gram kidney, with the uranium concentration remaining above 3 µg per gram kidney for a few weeks (see Annexe A, Section A3.2). Renal effects were observed 78 days after the accident and indicators of kidney function remained abnormal for 455 days post-exposure. The peak concentration of uranium in the kidney was much lower in this case than in the case described by Pavlakis et al (1996), and in the case of skin exposure described by Zhao and Zhao (1990), which is consistent with the less severe effects on kidney function.

Butterworth (1955) reported another case of dermal exposure to hot uranium compounds. In this case the predicted maximum kidney concentration was about 3µg uranium per gram ten days after the accident, with the level remaining above 1µg per gram for 20-30 days (Annexe A, Section A3.5). Some adverse effects on the kidney (albuminuria) persisted until the beginning of the third week after exposure. Butterworth (1955) also described an experiment in which a volunteer ingested 1 g uranyl nitrate which would lead to a maximum predicted kidney concentration of about 1 µg uranium per gram (Annexe A, Section A3.4). Albuminuria was observed only twice when uranium excretion was at its highest. Kidney dysfunction was also detected in some terminally ill patients receiving intravenous uranium intakes that are predicted to have lead to peak concentrations of about 1-6 µg uranium per gram kidney (Luessenhop et al 1958; Annexe A, Section A3.9). These studies show that effects on the kidney can be observed after acute intakes which transiently lead to levels of about 1 µg uranium per gram kidney.

1.4.3 Summary of toxic levels of uranium in humans

The suggestion that adverse effects on the kidney can be prevented if the concentration of uranium is maintained below 3 µg per gram kidney is still widely cited, although there are numerous studies with laboratory animals, and limited data from humans, that show that adverse effects on the kidney can be detected at kidney uranium concentrations that are very much lower than this. In susceptible animals, concentrations of uranium in the kidney as low as 0.02 µg per gram can have detectable effects on kidney morphology and severe effects have been observed in animals at concentrations of 3.5 µg per gram (Gilman et al 1998a).

In a review of the toxicity of uranium, Leggett (1989) has suggested that the occupational limit based on 3 µg uranium per gram kidney is about ten-fold too high. This view is consistent with the studies of Limson Zamora et al (1998), which suggest chronic intakes resulting in kidney concentrations of 0.1 µg uranium per gram can result in detectable kidney dysfunction, and the studies of acute exposures described above which indicate that transient effects on the kidney can occur at concentrations of about 1 µg uranium per gram kidney. The view that uranium might be more toxic than previously recognised has been accepted by the WHO which has proposed cautious chronic exposure limits for

Table 1.1. Chronic human exposures to uranium resulting in effects on the kidney

Intake route	Chemical form	Subjects	µg U per gram kidney	Effect	Reference
Inhalation	Yellowcake	27	up to ~1	++	Thun et al 1985
Intramuscular	Uranium metal	15	up to ~0.5	–	Hooper et al 1999
Ingestion	Drinking water	30	up to ~0.1	++	Limson Zamora et al 1998

Biochemical indicators of renal dysfunction: ++ Protracted – Negative
It should be noted that the investigations of renal function have greatly improved over the last 40 years, therefore subtle effects on renal function may not have been noted in the older references.

Table 1.2. Acute human exposures to uranium resulting in effects on the kidney

Intake route	Chemical form	Subjects	Intake, mg U	µg U per gram kidney	Effect	Reference
Ingestion	Acetate	1	8500	100	+++	Pavlakis et al 1996
Dermal (burn)	Nitrate	1	130	35	+++	Zhao and Zhao 1990
Inhalation	Tetrafluoride UF_4	1	900	10	++	Zhao and Zhao 1990
Injection	Nitrate	2	10	5	++	Luessenhop et al 1958
Dermal (burn)	Nitrate	1	10	3	++	Butterworth 1955
Inhalation	Ore concentrate	1	200	3	–	Boback 1975
Injection	Nitrate	3	5	2	+	Luessenhop et al 1958
Inhalation	Hexafluoride UF_6	3	50–100	1–3	+	Kathren and Moore 1986
Ingestion	Nitrate	1	470	1	+	Butterworth 1955
Inhalation	Hexafluoride UF_6	1	20	1	–	Boback 1975

Severe clinical symptoms +++
Biochemical indicators of renal dysfunction: ++ Protracted + Transient – Negative
It should be noted that the investigations of renal function have greatly improved over the last 40 years, therefore subtle effects on renal function may not have been noted in the older references.

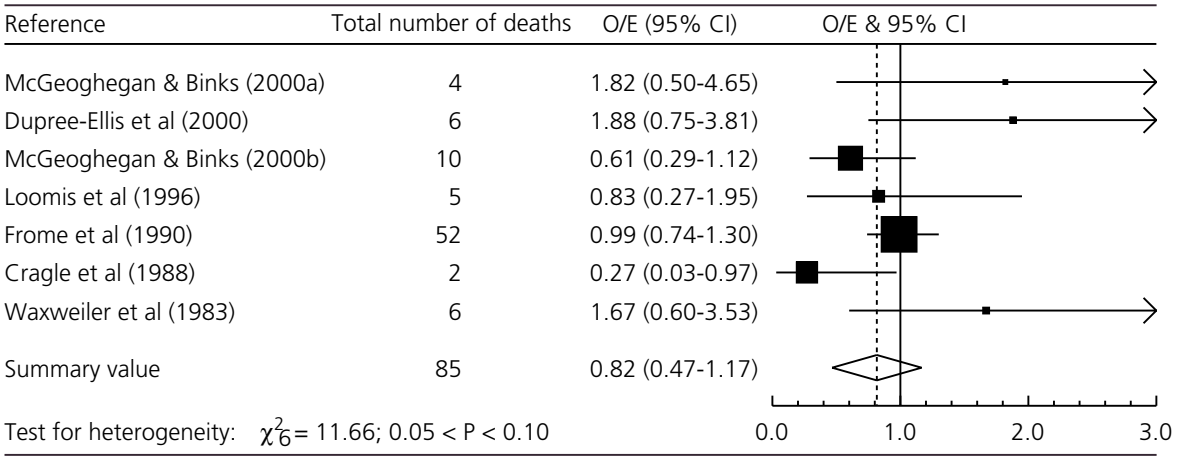
the general public based on one-hundredth of those intakes that result in slight adverse kidney effects in animals. The WHO tolerable daily intakes of 0.5 µg per kg body mass per day for ingestion of soluble uranium compounds, and 5 µg per kg body mass per day for insoluble compounds, should maintain kidney uranium concentrations below 0.01 µg per gram. Similarly, the proposed limits of 0.5 µg per kg per day for inhaled soluble or insoluble uranium should also maintain kidney uranium concentrations below 0.01 µg per gram. A summary of chronic human exposures to uranium resulting in effects on the kidney is given in table 1.1.

Acute intakes somewhat above these proposed limits for the general public are likely to be well tolerated but the kidney uranium concentrations that result in a significant increase in the probability of kidney disease in later life are very poorly understood. There is a better understanding of the levels of uranium that produce acute toxic effects on the human kidney. The studies of humans exposed to large intakes of uranium indicate that concentrations of over about 50 µg uranium per gram kidney are likely to lead to acute kidney failure that would be lethal in the absence of appropriate medical intervention. Thus, in the acute exposures described above, the patient who had an estimated peak level of about 100 µg uranium per gram kidney was in a critical condition requiring dialysis, and the patient with a peak level of about 35 µg per gram was in a serious condition (although burns contributed to his condition), whereas the patient in which the level was estimated to reach 10 µg per gram was much less severely ill. The kidney is a resilient organ and the individuals receiving these large intakes recovered adequate kidney function, although since the publication of these reports there has been no further information on their health so the long-term consequences of their uranium-induced kidney damage are unknown. A summary of the acute human exposures to uranium resulting in effects on the kidney is given in table 1.2.

1.5 Kidney disease in uranium workers

Inhalation of uranium dust occurs during mining and milling of uranium ores, in the processing of ores into uranium metal and during the conversion of processed uranium into fabricated metal products. Many epidemiological studies have been carried out on the health of workers in the mines and industrial plants carrying out these activities (see Part I and NECIWG 2000). Such studies are problematic as exposures to

Figure 1.3. Ratio of observed number of deaths from chronic renal failure in uranium workers compared to that expected in the general population.



many other toxic materials occur in all of these settings. These include other radioactive materials (eg radon in uranium mines), other toxic heavy metals (eg cadmium, vanadium and lead), silicates, diesel exhaust, and large quantities of chemicals, solvents and degreasers. It has been suggested that the toxic hazards from chemicals and solvents in some processing and fabrication plants may exceed the radiation hazards (NECIWG 2000). Thus, even if an increased death rate from malignant or non-malignant disease could be established among industrial workers handling uranium, it would be difficult to link this with certainty to uranium exposure rather than to exposure to other toxic materials.

There are also considerable problems in establishing whether the number of observed deaths from all causes, or from any specific causes, are greater than they would have been in the absence of occupational exposure to uranium. A general problem is the healthy worker effect, where those employed by the uranium industry are likely to be more healthy than the general population. In the absence of any occupational risks, the uranium workers would be expected to have slightly lower death rates from malignant and non-malignant disease than the general public. Furthermore, even in large cohorts, small differences between death rates in uranium workers and the general public will occur simply by chance.

Epidemiological studies of malignant disease in uranium workers have been reviewed in Part I of this report. The main concern from the chemical toxicity of uranium is the effect on the kidney. There are relatively few studies that examine deaths from kidney disease in industrial settings where uranium is handled and even fewer on morbidity rather than mortality.

In the epidemiological studies reviewed in Part I there were 151 deaths from kidney cancer among the 120,000 uranium workers, which was 22% fewer than the expected number of deaths in the general population (see table 6, and also Appendix 3, and figure 10 of Annexe I, Part I). There were very few deaths from kidney cancer in eight of the nine studies that recorded deaths from this cause. In four of these studies there were more deaths from kidney cancer than expected, but the number of deaths in these studies was very small (eight or fewer), and none of the excesses were statistically significant. The one study that was large enough to include a substantial number of deaths from kidney cancer was the combined study of workers at Oak Ridge (Frome et al 1997). The 109 deaths from kidney cancer among these workers were slightly fewer than expected.

In the same studies, although there were over 300 deaths from genitourinary diseases (mainly kidney disease), this was 30% fewer than the expected number from genitourinary disease mortality rates in the general population (see table 6, and also Appendix 3, and figure 17 of Annexe I, Part I). Furthermore, in every study the number of deaths observed was fewer than the number expected in the general population, although most of these studies included few deaths from this cause. The only report where a substantial number of deaths from genitourinary disease occurred was the large combined study of workers from four nuclear plants at Oak Ridge, Tennessee (Frome et al 1997). In this study there were 270 deaths from genitourinary disease, which was significantly fewer than the number expected.

Seven studies also examined deaths specifically from chronic renal failure (figure 1.3).

Overall there were 85 deaths, which was 18% fewer than the number expected from mortality rates in the general population. In three studies the number of deaths observed was greater than the number expected. However, these studies included no more than six deaths each and in no case was the excess significant statistically. In the largest study, which included 52 deaths, the ratio of observed to expected deaths was 0.99.

Table 1.3. Summary of predicted maximum concentrations of uranium in the kidney following DU intakes estimated for various scenarios. Values greater than or equal to 3 µg uranium per gram kidney are highlighted in bold as this level has often been used as a basis for occupational exposure limits.

Scenario	Central estimate (µg per gram kidney)	Worst-case (µg per gram kidney)
Level I inhalation of impact aerosol	**4**	**400**
Level II inhalation of resuspension aerosol within contaminated vehicle	0.05	**96**
Level II ingestion within contaminated vehicle	0.003	**3**
Level III inhalation of resuspension aerosol within contaminated vehicle	0.005	**10**
Level III ingestion within contaminated vehicle	0.0003	0.3
Level III inhalation of plume from impacts	0.0009	0.6
Level III inhalation of plume from fires	0.00012	0.05
Level III inhalation of resuspension from ground	0.003	**4**

There is some evidence that chronic renal failure is elevated in some groups of uranium miners (Thun et al 1982; BEIR IV 1988), but these workers are exposed to radon and typically also to a number of other toxic compounds, and the cause of the excess may not be the chemical toxicity of uranium. There is therefore no clear evidence that occupational exposure to uranium results in increased deaths from kidney cancer or chronic renal failure.

Large epidemiological studies examine cohorts of workers that have very variable levels of exposure to uranium, usually without any quantitative measures of exposure, and thus increases in mortality among small groups of workers with high levels of exposure may be obscured. Some studies have been able specifically to address the health of those workers who are likely to be most heavily exposed to uranium. One study has investigated both malignant and non-malignant causes of death in workers involved in the milling of uranium ore (Waxweiler et al 1983). In this study there were three deaths from kidney cancer compared with 2.7 expected, and six deaths from chronic renal failure compared with 3.6 expected. Neither of these increases is significant statistically.

Although there is no clear evidence that increased deaths have occurred due to elevated levels of uranium in the kidneys of uranium workers, there is some evidence of reduced kidney function (Thun et al 1985; see Section 4.1.2).

1.6 Uranium toxicity and DU munitions

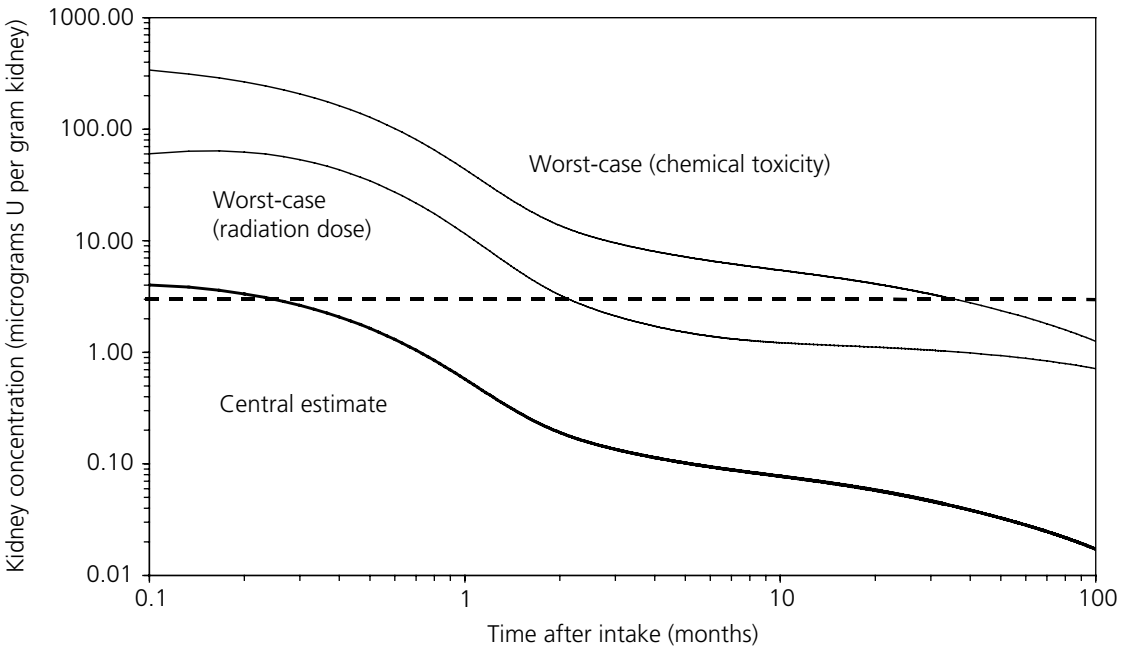
1.6.1 Kidney effects from intakes of DU on the battlefield

Exposures from the military use of DU will mostly occur by inhalation of impact aerosols and by inhalation and ingestion of DU from contaminated surfaces. Exposure to DU resulting from the solubilisation of DU shrapnel in some soldiers has also to be considered. The estimated maximum concentrations of uranium in the kidneys for different battlefield scenarios are given in table 1.3. An explanation of the exposure scenarios is given in Part I of the report (Section 2.2). In correspondence with veterans it was pointed out that some staff of medical field units in the Gulf War would have been exposed to DU dust from the contaminated clothing of allied or Iraqi casualties. Some of these medical personnel could be considered to have received Level II or Level III exposures to DU, depending on the total time they were exposed to inhalation intakes of DU dust while removing or handling contaminated clothing.

We have made two assessments of kidney concentrations for each scenario:

- A '*central estimate*', intended to be a central, representative value, based on the likely values of relevant parameters (intakes of DU, solubility of DU oxides, etc) that determine the amount of uranium reaching the kidneys according to the information available, or where information is lacking, values that are unlikely to underestimate the levels greatly. The central estimate is intended to be representative of the average individual within the group (or population) of people exposed in that situation.
- For individuals in each group levels could be greater than (or less than) the central estimate. We calculated a '*worst-case*' estimate using values of the relevant parameters at the upper end of the likely range, but not extreme theoretical possibilities. The aim is that it is unlikely that the uranium level in the kidney for any individual would exceed the worst-case. Thus the worst-case should not be applied to the whole group to estimate, for example, the number of individuals who might have kidney damage. One aim of the worst-case assessments is to try to prioritise further investigation. If even the

Figure 1.4. Predicted concentration of uranium in kidneys following an estimated Level I inhalation intake of DU oxide. Acute intakes of 250 mg (central estimate) or 5000 mg (worst-case), and the parameter values from Part I, Appendix 1, table 14, are used. The levels of uranium in the kidney are shown for the central estimate, for the worst-case for chemical toxicity and for radiation dose; uranium levels in the kidney are less under the conditions that maximise the radiation dose. The bold horizontal broken line indicates a concentration of 3 µg uranium per gram of kidney.



worst-case assessment for a scenario leads to low levels of uranium in the kidney, then there is little need to investigate it more closely. If, however, the worst-case assessment for a scenario leads to significant levels, it does not necessarily mean that such high levels have occurred, or are likely to occur on a future battlefield, but that they might have occurred, or might occur in future conflicts, and further information and assessment are needed.

Details of the methods used and assumptions made in estimating the intakes of DU are provided in Part I, Appendix 1.

1.6.2 Kidney effects from central estimates of intakes

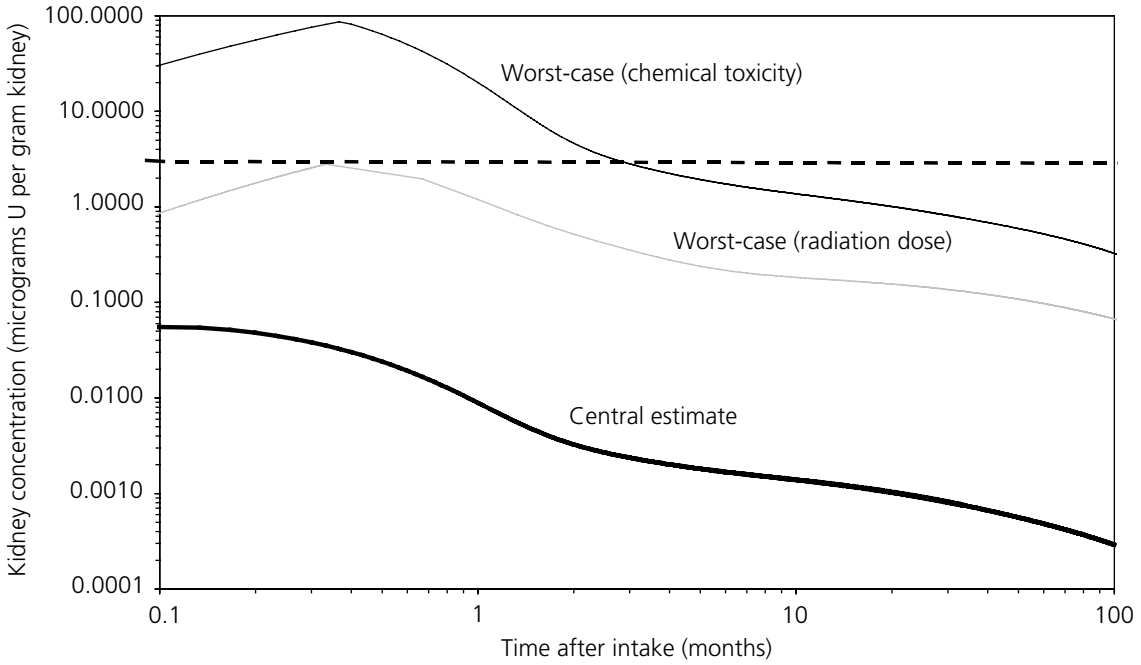
For the central estimates, the maximum concentrations of uranium in the kidney for the Level II ingestion scenario, and all Level III scenarios, are predicted to be less than or equal to 0.005 µg per gram kidney. It is highly improbable that the peak uranium concentrations in the kidney achieved under the central estimate assumptions for these scenarios will lead to any significant effects on kidney function. The estimated maximum kidney concentration from the Level II inhalation exposure (0.05 µg per gram kidney) is slightly greater than the kidney uranium concentration in rabbits at chronic intakes that produced slight effects on the kidney (0.02-0.04 µg per gram kidney), and is about seven times greater than the kidney concentration estimated for the WHO tolerable daily intake. However, a kidney uranium concentration that transiently reaches a maximum of 0.05 µg uranium per gram is also unlikely to produce any long-term adverse effects on the kidney.

The central estimate for the Level I inhalation scenario predicts a peak kidney uranium concentration of about 4 µg per gram. From the limited information available on the toxicity of uranium in humans it is considered that a concentration of 4 µg uranium per gram of kidney for about a week (figure 1.4) is likely to cause some damage to the kidney. Kidney function can be reduced by as much as two-thirds without any obvious symptoms, and soldiers exposed to DU intakes that transiently result in concentrations as high as 4 µg uranium per gram of kidney are unlikely to show any clinical signs of kidney dysfunction, although some dysfunction could well be apparent for a short period after the intake using biochemical markers of kidney function. Whether such an exposure would lead to any long-term effects or would increase the chance of kidney disease in later life is unknown, but we consider it unlikely.

1.6.3 Kidney effects from worst-case estimates of intakes

The worst-case peak concentration of uranium in the kidney arising from Level I inhalation exposures to DU is very high (about 400 µg uranium per gram kidney). This level greatly exceeds the occupational limit of 3 µg uranium per gram kidney, which is believed to be set at too high a level, and would result in uranium concentrations in the kidney above this occupational

Figure 1.5. Predicted concentration of uranium in kidneys following an estimated Level II inhalation intake of DU oxide. Acute intakes of 10 mg (central estimate) or 2000 mg (worst-case), with parameter values from table 15 of Part I, Appendix 1, are used. The levels of uranium in the kidney are shown for the central estimate, for the worst-case for chemical toxicity and for radiation dose; uranium levels are less under the conditions that maximise the radiation dose. Note that the worst-case is based on 100 hours exposure at 20 mg intake per hour and is represented here by 10 intakes of 200 mg on 10 consecutive days. This results in a slightly lower maximum concentration (87 µg uranium per gram kidney), than a single intake of 2000 mg (96 µg uranium per gram kidney: table 1.3). The bold horizontal broken line indicates a concentration of 3µg uranium per gram kidney.



limit for a few years even supposing normal kidney function were maintained (figure 1.4). A very high peak kidney concentration (about 100 µg uranium per gram kidney) is also predicted for the worst-case Level II inhalation exposure and the level would remain above 3 µg per gram for several months (figure 1.5).

These estimated worst-case peak kidney uranium concentrations are substantially higher (Level I inhalation exposure), or as high (Level II inhalation exposure), as the peak kidney uranium concentrations predicted to have occurred in all of the cases of accidental exposures to uranium, where very severe effects on the kidney were observed. It therefore seems likely that the worst-case estimates of the amounts of DU reaching the kidneys after Level I or Level II inhalation exposures would lead to acute kidney failure that would be lethal in the absence of appropriate medical intervention. It is not clear whether our worst-case kidney uranium levels would occur after intakes of DU on the battlefield, as they assume the highest estimates of intakes for each scenario and the values of the important parameters of the biokinetic models (particle size, solubility, etc) that maximise the amount of uranium reaching the kidney. If they did occur they would be expected to apply only to a small number of those soldiers receiving Level I or Level II inhalation exposures, and should be very apparent as they would be expected to result in acute distress and kidney failure soon after exposure.

The worst-case estimates for kidney damage will not be the worst-case for radiological effects on the lung. Although the intakes of DU are the same, the worst-case for radiological damage to the lung assumes the lowest observed values for the solubility of DU particles, whereas the worst-case for kidney damage assumes the highest observed values for solubility. An individual with the worst-case estimate for lung cancer would therefore not have the worst-case risk of kidney damage and *vice versa* (see figures 1.4 and 1.5).

The worst-case Level III inhalation scenario (inhalation of DU oxide dust resuspended in the air as a result of briefly entering contaminated vehicles and disturbing dust on the inside surfaces) is also predicted to give a high peak kidney uranium concentration (10 µg per gram) and this level may lead to some significant kidney damage. The long-term consequences of this level of uranium in the kidney are unclear. A peak concentration of 3 µg per gram is estimated for the worst-case Level II ingestion of DU within a contaminated vehicle, and 4 µg per gram for Level III inhalation of DU oxide dust that has been deposited on the ground and subsequently 'resuspended' in the air as a result of disturbance by wind, vehicle movements, etc. These levels may also lead to some minor short-term kidney damage, although long-term effects are considered unlikely.

Table 1.5. Predicted maximum concentrations of uranium in the kidney following long-term DU intakes from resuspended soil.

Scenario	Central estimate (µg per gram kidney)	Worst-case (µg per gram kidney)
Long-term inhalation of resuspension from ground:		
Adult	0.002	0.2
Ten year-old child	0.001	0.1
One year-old child	0.001	0.1

1.6.4 Kidney effects from retained DU shrapnel

The average kidney uranium concentration estimated for the veterans with retained DU shrapnel (0.1 µg uranium per gram kidney) is similar to that at which slight effects on the human kidney were observed using sensitive tests of kidney function by Limson Zamora et al (1998). However, no clinical or biochemical signs of kidney dysfunction have been reported in any of these veterans (McDiarmid et al 1999, 2000, 2001; McDiarmid 2001; McClain et al 2001), which is somewhat surprising as the highest level of kidney uranium (0.5 µg uranium per gram kidney) is estimated to be about four times that at which effects were observed by Limson Zamora et al (1998).

Chronically elevated levels of uranium in the kidney might be expected to lead to greater effects on the kidney than those that arise from acute exposures which transiently lead to the same elevated levels of uranium. However, there is evidence from animal studies that chronic exposure leads to an increased tolerance to the nephrotoxic effects of uranium (Leggett 1989). This effect was apparent in rats with implants of DU pellets where no histological or functional signs of kidney damage were apparent, although the measured levels of uranium in the kidney were greater than those that are known to be nephrotoxic after acute intakes (Pellmar et al 1999a). The lack of any signs of kidney dysfunction in the soldiers with retained DU shrapnel needs to be treated with caution as animal studies indicate that apparent tolerance to uranium still results in alterations of kidney histology (Leggett 1989), and an increased chance of kidney dysfunction in later life among these veterans cannot be ruled out.

The possible consequences of the radiation from the retained fragments of DU have been discussed in the first part of the report, as has evidence from animal studies that uranium might act directly to damage the genetic material of cells (see Part I, Appendix 2). Cells surrounding retained DU shrapnel (or particles of DU in the lung or associated lymph nodes) will be bathed in a high local concentration of uranium and the damaging effects from irradiation could be enhanced by direct chemical effects on the genetic material from the uranium. It should be stressed that there is no evidence that this occurs, but it is a concern and an area where there are ongoing experimental studies with laboratory animals.

1.6.5 Kidney effects from long-term intakes of DU

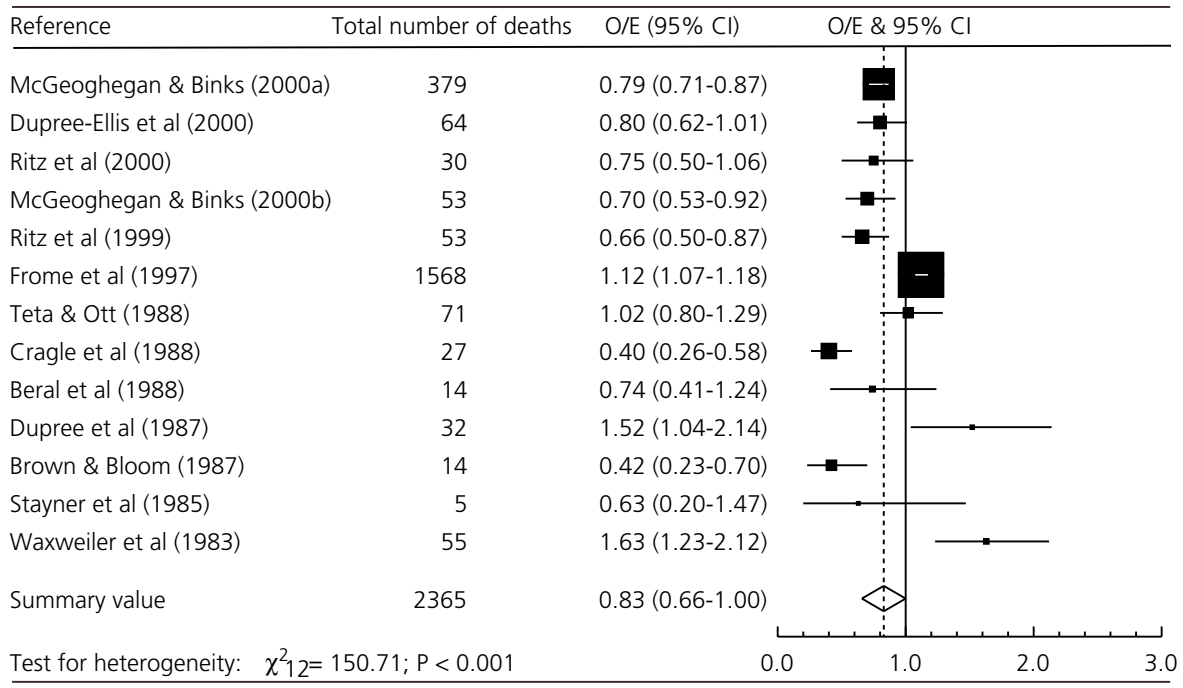
Adults and children returning to live in areas where DU munitions were deployed may be chronically exposed to slightly elevated levels of uranium by inhalation of DU particles from resuspended soil and by ingestion of contaminated food and water (see Chapter 2). For children and adults the central estimates of kidney uranium concentrations from the long-term inhalation exposures to DU are predicted to be at least five-fold less than the kidney uranium concentration at the WHO tolerable daily intake (table 1.4; see Annexe F for calculations).

Worst-case estimates of the kidney uranium concentrations from long-term inhalation exposures for adults and children returning to areas where DU munitions were deployed are predicted to be 0.1-0.2 µg per gram (table 1.4; see Annexe F). These chronic exposures would be expected to result in minor kidney dysfunction, as the kidney concentrations are greater than those where adverse effects were observed in the study of individuals chronically exposed to elevated levels of uranium from some private water sources (Limson Zamora et al 1998). It should be remembered that the worst-case estimates would be expected to apply to only a small number of individuals, if any.

The increased risk of cancer from inhalation of resuspended DU particles will be very small for both children and adults. The greatest risk is to the lung, but even the worst-case excess risk of fatal lung cancer is only about 6 per 100,000; the central estimate is 100-fold lower (see Chapter 2). There are however substantial uncertainties in estimating central or worst-case inhalation intakes of DU in the years following a battle (Part I, Annexe F).

Estimates of intakes of DU from contaminated food or water, or from ingestion of soil, are very difficult to make and have not been attempted, but are likely to be highly variable (see Chapter 2).

Figure 1.6. Ratio of observed number of deaths from non-malignant respiratory disease in uranium workers compared to that expected in the general population.



1.7 Other non-malignant effects of uranium

1.7.1 Bone effects

Uranium accumulates in bone, which is thus considered a tissue at risk from the toxicity of large acute or chronic exposures to uranium. In the rat, both acute and chronic intakes cause a decrease in bone formation and may increase bone resorption (Ubios et al 1991). There is very little information on the effects of uranium on bone formation or strength in humans. It is therefore difficult to evaluate whether effects on bone are expected in those who have received large intakes of DU.

1.7.2 Immunological effects

In Part I of the report the radiological effects of exposure to DU were examined but these were restricted to effects on the incidence of cancer. At the public meeting it was suggested that we should examine whether radiation from internalised DU might have adverse effects on the immune system. Although Part II of the report focuses on the chemical toxicity of uranium, the possibility of radiological effects on the immune system is considered in Chapter 3.

1.7.3 Neurocognitive effects

Elevated uranium concentrations have been shown to be present in the hippocampus region of the brains (an area associated with memory and learning) of rats implanted with DU pellets and have been associated with slight alterations of the electrophysiology of the brain (Pellmar et al 1999b). A statistical relationship has been observed between uranium levels in the urine of US Gulf War veterans and poorer results in computerised tests that assessed performance efficiency, but effects on cognitive ability were not observed (McDiarmid et al 2000). Possible effects of stress and anxiety resulting from their wounds and exposure to DU are difficult to rule out. Neurological and psychological problems are increased among Gulf War veterans (Cherry et al 2001a), but it is not possible to conclude whether this may be linked in any way to their exposure to DU or to any of the other potentially toxic exposures in the Gulf War.

1.7.4 Respiratory disease

Workers in the uranium industry and underground uranium miners have been chronically exposed to uranium dusts but there are few data on rates of non-fatal respiratory disease. Deaths from non-malignant respiratory diseases in uranium workers (excluding underground miners) are summarized in figure 1.6.

Overall the number of deaths observed in the combined studies was 17% fewer than the number expected from general population rates, although in three individual studies (Waxweiler et al 1983; Dupree et al 1987; Frome et al 1997) the numbers of deaths observed were significantly greater than the number expected from general population rates, by factors of 1.12, 1.52 and 1.63, respectively. Some studies therefore suggest a significant increase in mortality from non-malignant respiratory disease among uranium workers (NECIWG 2000), but in interpreting these results it must be remembered that mortality from many respiratory diseases (eg chronic bronchitis) is determined largely by smoking habits, and other toxic exposures may be present. However, the findings do rule out the possibility of large increases in respiratory deaths among uranium workers.

Occupational exposure to a number of metal dusts or fumes has been associated with several non-malignant lung diseases (Nemery 1990; Kelleher et al 2000). However, uranium is not one of the metals that have been clearly associated with these types of lung disease.

Scarring and thickening of lung tissue leading to shortness of breath and eventual cardiac failure has been observed in uranium miners but has been attributed to alpha-particles from highly radioactive radon progeny and possibly silicates (Archer et al 1998).

Pulmonary damage has also been observed in animals after long-term inhalation of some uranium compounds at concentrations above about 5 mg per cubic metre (Leach et al 1973; Spoor and Hursh 1973). Effects on the lung, including pneumonitis progressing to fibrosis and eventual death, have been observed in dogs following inhalation of aerosols of plutonium oxide, a highly radioactive alpha-emitter (Muggenburg et al 1988, 1999). These effects occurred at radiation doses to the lungs that were higher than, but of the same order of magnitude as, the lung doses from DU in the worst-case Level I intakes.

Some soldiers on the battlefield may receive inhalation intakes of DU oxides that are very substantially greater than the daily intakes that occur in chronically exposed uranium workers and the increased risks of lung cancer in such soldiers have been considered (see Part I). The nature of the inhalation intakes (particle size, presence of a significant ultrafine component, solubility, etc) are also likely to be different in the industrial setting (and in animal experiments) compared with the battlefield, which increases the difficulty in assessing the respiratory toxicity of inhaled DU. Acute respiratory effects would not be unexpected following the inhalation of large amounts of dense DU aerosols (for example, for any survivors in a tank struck by a DU penetrator or those working for protracted periods in contaminated vehicles).

It is unclear whether large inhalation intakes of DU would lead to sufficient alpha-particle irradiation of the lung to cause significant fibrosis, but the possibility perhaps exists for worst-case Level I or II intakes as the radiation doses are not very much lower than those at which pulmonary effects occur in dogs, and there is evidence that dogs may be about two-fold less sensitive to radiation-induced pulmonary damage than humans (Poulson et al 2000).

Long-term respiratory effects for soldiers who inhaled smaller amounts of DU from aerosols (most Level II and all Level III inhalation exposures) are considered unlikely.

1.7.5 Effects on reproductive health

Pellmar et al (1999a) reported significant levels of uranium in the testicles of rats implanted with DU pellets. Uranium has been shown to be present in the semen of veterans retaining fragments of DU shrapnel and presumably would be present in the semen of soldiers heavily exposed to DU aerosols. This raises the possibility of adverse effects on the sperm from either the alpha-particles emanating from DU, chemical effects of uranium on the genetic material (Miller et al 1998a,b) or the chemical toxicity of uranium. Synergistic effects from the combination of both radiation damage and direct chemical damage to the genetic material are also possible (See Part I, Appendix 2).

Studies on the reproductive health of workers in the nuclear industry, and of survivors of the atomic bombs, show little evidence of decreased fertility, or of an increased incidence of miscarriages or birth defects (Otake et al 1990; Doyle et al 2000). For example, a large study of over 20,000 pregnancies in the partners of male radiation workers at the Atomic Weapons Establishment, the Atomic Energy Authority and British Nuclear Fuels who had been exposed to radiation prior to conception showed no increase in foetal deaths or malformations. The lack of effect was seen both for workers who were only monitored for external radiation and for those monitored for both internal and external radiation. A slight increase in early miscarriages and stillbirths was found in pregnancies involving women radiation workers exposed prior to conception, but its significance is unclear as there was little evidence that the effect increased with radiation dose (Doyle et al 2000).

Effects of uranium on reproductive health have been observed in male mice, although at very high intakes. Daily ingestion of large amounts of soluble uranium (between 10 and 80 mg uranium per kg per day; equivalent to 700 mg to 5.6 g per day for a 70 kg man) over nine weeks had no apparent effect on testicular function or sperm development, but there were some effects on the morphology of the hormone-producing cells in the testes at the highest exposure level. A decrease in male fertility was reported but this was not related to the level of uranium exposure and its significance is unclear (Llobet et al 1991).

In other studies, the offspring of male mice injected with plutonium-239 (a highly radioactive alpha-emitter) showed an increased predisposition to the induction of leukaemia by a chemical mutagen (Lord et al 1998), but the intake that would be required to produce the same dose to the testes of a 70 kg man using the much less radioactive DU would be far above that causing lethality due to the chemical toxicity of uranium. We are not aware of any animal studies that have looked for developmental abnormalities in the progeny of uranium-exposed males.

Uranium is known to cross the placenta (Sikov and Mahlum 1968; McClain et al 2001) and increased levels

of uranium in the mother will lead to increased levels in the foetus. The effects of exposure of pregnant mice to uranium have been studied by Domingo et al (1989a). Ingestion of 5 mg of soluble uranium per kg per day during pregnancy had no effect on sex ratios, mean litter size, body weight or body length of the newborn mice at birth or during the subsequent three weeks. Exposure of male mice to ingested soluble uranium for two months prior to mating with females that were also exposed prior to and during pregnancy resulted in some embryo lethality at intakes of 25 mg per kg body weight (Paternain et al 1989). Doses of 5 to 50 mg of soluble uranium per kg per day in food during pregnancy have been shown to reduce foetal body weight and body length, and to produce developmental defects including cleft palate and skeletal abnormalities (Domingo et al 1989b). These effects were particularly apparent at the 25 and 50 mg per kg dosages but some effects were apparent at 5 mg per kg. Developmental effects and malformations were also observed in mice born to mothers given daily subcutaneous injections that resulted in severe maternal toxic effects including death (Bosque et al 1993). The significance of these effects in mice is unclear as they occur at high intakes of soluble uranium that are equivalent to between 250 mg and 2.5 g per day for a 50 kg (eight stone) woman.

There are uncertainties in extrapolating from animal studies to humans and there is a possibility of effects on reproductive health for soldiers who have high levels of exposure to DU, and careful epidemiological studies are required. An important study of the reproductive health of male and female UK Gulf War veterans and the health of their children has been carried out by Dr Pat Doyle and colleagues, although the results of the study are not yet available. The study compares soldiers who served in the Gulf with a similar group of military personnel who were not deployed in the Gulf. The adverse endpoints being examined include infertility, foetal loss, low birth weight, congenital malformation and childhood illness. If there is a significant effect on reproductive health it will be difficult to establish whether this is due to DU or to any of the other potentially toxic exposures in the Gulf War.

There are reports in the media and elsewhere of increased rates of foetal death and malformations in children born in Iraq and Bosnia since the conflicts in these regions. These reports are of obvious concern but are very difficult to interpret as reliable data on the rates of foetal death and malformation prior to and following these conflicts are not available. Recently, the WHO has initiated studies to ascertain whether reproductive health in Iraq has declined since the Gulf War. If there have been increased rates of foetal death and malformation it will again be difficult to know whether this is due to DU as the population of Iraq has been subjected to multiple toxic exposures.

It should also be remembered that malnutrition can increase the incidence of malformations (eg the link between neural tube defects and folic acid deficiency is firmly established), and a deteriorating quality of food supplies and storage conditions can increase exposure to mycotoxins which are potent teratogens.

1.8 Conclusions

Uranium is a poisonous metal with its most toxic effects being exerted on the kidney. The levels of uranium in the human kidney that cause kidney damage, and the long-term effects of acute and chronic intakes of uranium are not well understood. Numerous studies with animals have been carried out but these show substantial differences in the lowest kidney uranium concentrations that result in adverse effects. In some studies with rabbits, chronic ingestion leading to kidney uranium concentrations as low as 0.02 µg per gram of kidney has observable effects on kidney morphology, whereas studies with rats indicate that concentrations as high as 0.7 µg per gram kidney have little effect. Current exposure limits for chronic ingestion of uranium for the general public have used the lowest chronic intakes that result in adverse effects on the kidneys of rabbits (Gilman et al 1998a) - ingestion of 50 µg soluble uranium per kg body mass per day - and have reduced this intake by a factor of 100 to take into account the uncertainties in extrapolating from rabbits to humans. Chronic ingestion of soluble uranium below this limit (0.5 µg per kg per day) should result in a kidney uranium concentration below 0.01 µg per gram of kidney. The tolerable daily intakes of uranium by inhalation are also expected to maintain the kidney uranium concentrations below this level.

The limited data on human exposures support the view that the level of 3 µg uranium per gram kidney proposed as a basis for occupational exposure limits is too high. Although the concentrations which produce toxic effects on the human kidney are poorly understood, most of the data are consistent with the view that adverse effects in humans can be detected at chronic intakes that result in kidney concentrations of about 0.1-0.5 µg uranium per gram, or acute intakes resulting in about 1 µg per gram, but the long-term effects (if any) of these elevated uranium levels are not clear.

The studies of human exposures that are of most relevance to the intakes of DU that occur on the battlefield are the small number of case reports that describe the effects of large acute intakes of uranium. These studies suggest that acute intakes predicted to result in peak concentrations of greater than 50 µg uranium per gram kidney are likely to result in very serious effects on the kidney that may be lethal in the absence of appropriate medical intervention. However,

this conclusion is based on a very few cases of large acute exposures. The kidney is a resilient organ and even individuals who have received these high intakes of uranium appear to recover kidney function, although some abnormalities may remain detectable for several years. The long-term effects of acute uranium poisoning in humans are not known but clearly could lead to an increased likelihood of kidney failure in later life.

Similarly, the long-term consequences of transient exposures to lower levels of uranium in the kidney are poorly understood. It is not possible to estimate with any confidence how long uranium concentrations that lead to slight biochemical signs of kidney dysfunction can be tolerated in humans, or how far above this threshold concentration exposures can be without long-term adverse effects on the kidney.

Epidemiological studies provide little evidence for increased rates of kidney disease in uranium workers, but the absence of reliable data on the levels of uranium in the kidney makes it difficult to estimate exposures to uranium that lead to no significant increase in mortality from kidney disease. There are few data on non-fatal kidney disease in uranium workers and conflicting evidence from post-mortem examination of the kidneys of uranium workers. Effects on kidney morphology have been observed in some studies but not in others. However, inhalation intakes of uranium particles in industrial settings are chronic and, even before the introduction of stringent occupational safety standards, the daily intakes were probably much lower than the acute intakes that could be received under worst-case assumptions by some soldiers. Furthermore, the forms of the inhaled particles in industrial settings will typically be different from those on the battlefield, and these differences might lead to significant differences in their ability to lead to adverse effects.

The central estimates of kidney uranium concentrations in all exposure scenarios on the battlefield are unlikely to cause acute kidney problems, although for Level I exposures, and to a lesser extent Level II inhalation exposures, the possibility of minor kidney damage exists. The worst-case Level I and Level II inhalation scenarios are expected to lead to very severe acute effects on the kidney. It is not clear whether such exposures to DU would occur on a battlefield, but the occurrence of acute kidney problems, requiring hospitalisation and critical care within a few days or weeks of DU exposure, would indicate that soldiers might have received intakes that lead to very high levels of kidney uranium. The toxic effects of DU from these worst-case scenarios should therefore be much easier to observe that the worst-case radiological effects, as the effects on the kidney are rapid and obvious, whereas the development of lung cancer will typically take several decades. It should be stressed that the worst-case estimates for kidney damage will not be the worst-case for radiological effects. An individual with the worst-case estimate for lung cancer would therefore not have the worst-case risk of kidney damage and *vice versa*. However, for Level I inhalation exposures, the worst-case for radiological effects is still predicted to result in dangerously high peak kidney uranium concentration (about 50 µg per gram, compared with 400 µg per gram for worst-case chemical toxicity). For Level II inhalation exposures the peak kidney concentration would be much less under conditions which maximise radiation dose (about 3 µg per gram, compared with 96 µg per gram).

The fact that kidney function can be reduced by about two-thirds without any obvious symptoms, and the ability of the kidney to recover apparently normal function even after a large intake of uranium, has implications for the evaluation of the health of veterans. In the UK the Ministry of Defence Medical Assessment Programme for Gulf War Veterans recommends tests for uranium levels 'if the veteran has symptoms and signs that suggest such a test is clinically necessary'. This approach has no good scientific basis since several years after an exposure it is unlikely that any clinical signs (or perhaps even biochemical signs) of kidney dysfunction would be apparent, even in veterans who had been exposed to a large acute intake of DU. Any veterans who received intakes of DU that were substantial, but not large enough to cause acute symptoms of kidney damage, would not subsequently be identified so that their health (eg early signs of lung cancer) and kidney function could be followed. However, we should stress that, excepting Level I exposures, adverse effects on the kidney are not expected according to the central estimates of peak kidney uranium levels, although there might be significant kidney effects for some soldiers under the worst-case Level I and II assumptions. Long-term monitoring of kidney function using modern biochemical methods is recommended for any veterans who may have had substantial exposures to DU.

In animals, chronic exposure appears to lead to some tolerance to the nephrotoxic effects of uranium, which may explain the absence of signs of kidney dysfunction in veterans with retained DU shrapnel. The kidneys of animals with increased tolerance to uranium have been shown to have abnormalities (Leggett 1989) and the continuing surveillance of these veterans is required as kidney dysfunction in later life remains a possibility.

According to the central estimates, the long-term intakes of DU occurring after a conflict from resuspension of DU in soil are not expected to result in increased levels of kidney disease among returning civilians. Worst-case estimates of kidney uranium levels raise the possibility of some adverse effects on the kidney for inhalation intakes from resuspended DU.

Animal studies suggest that absorption of uranium from the gut of neonates might be higher than in older children or adults and that malnutrition could enhance the effect of uranium by increasing uptakes from the gastrointestinal tract to the blood. Malnutrition also can lead to ingestion of soil (geophagy), which if substantial could lead to significant intakes of uranium in DU-contaminated areas (Annexe C).

Short-term respiratory effects occurring soon after extremely large inhalation intakes of DU would not be surprising. Whether this would lead to any long-term respiratory effects is difficult to evaluate, but some fibrosis of the lung is perhaps possible if any soldiers received the worst-case Level I or II inhalation exposures.

Effects on immune function from the chemical effects of DU exposure or from internal radiation are considered unlikely. Exposure of the thoracic and extra-thoracic lymph nodes to alpha-radiation from retained particles of DU may lead to the killing of some immune cells traversing these lymph nodes but, in the absence of high doses to the red bone marrow, there is unlikely to be any measurable increase in susceptibility to infection, or other significant adverse immune effects, from the intakes of DU that could occur on the battlefield (see Chapter 3). The possibility of very slight effects which could exacerbate any adverse effects on the immune system from other toxic exposures present in modern warfare cannot be discounted.

There is inadequate information about the effects of elevated levels of exposure to uranium on human reproductive health. There is no evidence that male radiation workers in the uranium industry have suffered adverse effects on their reproductive health. However, uranium is known to cross the placenta and, in mice, high intakes of uranium by the mother have been shown to have effects on the foetus but these occur at very high intakes of soluble uranium that are toxic to the mother. Epidemiological studies of the reproductive health of Gulf War veterans and of the Iraqi population are underway, but if any adverse effects are observed it will be difficult to link them to DU, or to other potentially toxic exposures on the battlefield or other possible reasons.

2 Environmental impact of the use of DU munitions

2.1 Uranium in the environment

The health consequences arising from exposure to DU on the battlefield have been discussed in Part I of the report (radiological effects) and in Chapter 1 of this part of the report (chemical toxicity). The introduction of hundreds of tons of DU into the environment during battles where DU munitions are deployed may have longer term consequences for the health of those who continue to live in these areas and their environment. This part of the report discusses these environmental concerns and focuses on exposures to DU occurring in the years following conflicts where DU munitions were deployed. A more detailed account is given in Appendix 2 and the associated annexes. The intakes and risks for those living in conflict areas while DU munitions are being deployed will initially be similar to those of soldiers on the battlefield exposed to DU released from impacts and fires (Level III intakes from smoke plumes; Part I, Appendix 2, Section 8. 3). However, the exposure of the local residents to DU could continue for decades after a conflict as a result of environmental contamination.

Uranium occurs naturally within the environment and is widely dispersed in the earth's crust. Uranium is naturally present to varying extents in all rocks, soils, waters, atmospheric particles, plants and animals. The concentration of uranium in the soil and in plants and animals may be increased where uranium deposits occur close to the soil surface and uranium becomes mixed with the soil through weathering, or in areas in which uranium is artificially introduced. For example, soils that have developed over uranium-rich rocks such as granites generally contain higher concentrations of uranium compared with soils typically developed over sedimentary rocks. Once released from rocks, the uranium may then be dispersed into other parts of the environment, leading to naturally occurring uranium being widely dispersed.

Shortly after use, the main exposure of humans to DU on the battlefield is by inhalation and ingestion of the particles released from DU penetrators during impacts (or from shrapnel). However, people returning to, or continuing to live in, the battlefield will be exposed to DU from inhalation of DU particles resuspended from contaminated soil and dust, and from any contamination of water and food supplies. Exposure from inhalation of particles will reduce as DU is removed from the surface environment and, in the longer term, the environmental exposure pathways for DU become similar to the natural exposure routes where intakes of uranium from water or deliberate soil ingestion often dominate.

To determine the longer term environmental effects resulting from the use of DU munitions it is important to know the spatial distribution of the DU, where it came from, its physical and chemical form, and the extent to which different factors affect its movement in the environment. Only once these factors are known is it possible to compare the exposures to uranium from DU munitions with those from natural sources. The relative rates of environmental movement (migration) of uranium from DU penetrators in or on the ground, and from particles of DU oxides deposited on the ground from impacts, will determine the importance of the different routes by which various parts of the environment (such as groundwater, air, soil, plants and animals) might become contaminated.

Movement of DU into some components of the environment, such as water sources, may be very slow and take place over periods of time much longer than a human life. Consequently, contaminated land might be a concern for hundreds of years and environmental assessments need to take this into account; environmental monitoring carried out soon after a conflict might fail to find contamination of water supplies or other sensitive components of the environment and this might only become apparent after a number of years or more likely decades.

2.2 Environmental exposures to DU from military conflicts

Uranium has been mined and processed for use in nuclear reactors for several decades and, as a by-product of uranium processing, DU is plentiful and potentially cheap. Its high density makes it particularly useful as heavy-armour and kinetic energy penetrators. In these applications it is commonly alloyed with titanium that reduces its inherent tendency to corrode in moist air.

The chemical and mineralogical forms of DU released into the natural environment are difficult to characterise for every potential scenario. For example, in the case of military uses, the chemical form and amounts of the DU released into the environment will be heavily dependent upon the nature of the penetrator impact (ie the type and composition of the penetrator, the energy of impact and the composition of the impacted material) and any subsequent changes due to the DU coming into contact with soil or water.

2.3 DU in military conflicts

The nature and quantity of released DU has been reasonably well characterised during testing and on firing ranges (CHPPM 2000; Royal Society 2001). However, there are insufficient data to compare the composition and form of DU released under these controlled conditions with those under battlefield conditions. Since the first authenticated use of DU munitions was in the Persian Gulf War during 1991, there are very few data over environmentally significant timescales. For example, it is time periods greater than ten years, and more probably greater than 50 years, over which DU is likely to move significantly within the environment, leading to mixing with surface soils and groundwaters.

There are various estimates of the total amounts of DU used in the Gulf War and the Balkans. In the Gulf War, an estimate from data reported in CHPPM (2000) gives a total of about 339 tons. The quantity, form and location of DU released into the environment following military activities are related to the type and intensity of military action. Thus, large calibre tank rounds fired at armoured vehicles may often hit their targets causing large amounts of DU particles to be released, whereas in a strafing attack from an aircraft most of the smaller calibre penetrators will miss their target leaving many virtually intact penetrators buried in the ground. The environmental behaviour of DU particles released as impact aerosols will clearly be very different from that of the solid DU of intact penetrators that slowly corrode releasing uranium into the surrounding soil.

For the purposes of this report, the composition of DU released on the battlefield has been characterised by considering two groups: uranium-rich particles (dusts) generated during impacts and subsequent fires, and residual metallic fragments and nearly intact penetrators.

2.3.1 Uranium-rich dusts

Dusts containing mixed DU oxides can be generated during penetrator impacts and through the burning of DU-based materials. The two major factors that control the chemical and physical nature of these uranium-rich dusts are the force of impact and the composition of the impacted material. The amount of dust generated depends on the type of material the penetrator hits. For example, the most dust is considered to occur when a DU round penetrates a heavily-armoured vehicle, with much less release typically occurring following impact with softer targets or when DU rounds miss their targets. Preliminary data available from the Kosovo conflict suggest that dust production might be minimal during impacts between DU penetrators and concrete structures (MOD 2001; UNEP 2001). The corrosion/dissolution rates of such particles in the environment are relatively poorly studied compared with those in simulated biological fluids.

2.3.2 Residual metallic fragments and penetrators

The depth to which DU projectiles penetrate into soil depends on the mechanical and physical properties of the soil, and soil horizons (a layer of soil differing from adjacent layers in respect of colour, consistency, structure and texture in addition to chemical and biological differences). However, information on the relationship between penetration depth and soil characteristics has not yet been reported in the open literature. In Kosovo it has been considered that small calibre penetrators impacting into soft soil can penetrate the ground to a depth of up to 7 m with minimal production of DU dusts (UNEP 2001). In some cases in the Gulf War large calibre penetrators fired from tanks went through their target without oxidising or producing substantial quantities of dust, resulting in relatively large pieces of metallic DU entering the environment. These uncertainties, coupled with difficulties in identifying DU penetrators that have missed their target and become embedded in the soil, represent a significant knowledge gap, particularly where targets have been strafed and the proportion of penetrators hitting a hard target is low.

2.4 Corrosion and dissolution of DU

Corrosion is the general name given to a wide range of complex physical and chemical processes that result in detrimental changes to the fabric and structure of a given metal, and is similar in many ways to natural weathering processes. Metallic uranium or DU alloys can corrode through a number of processes, the majority of which are controlled by the local chemical environment in which the metallic uranium or uranium alloy resides. Corrosion can occur in air, water or water-containing soils. In addition to understanding the rate of corrosion, and the factors that alter the rate, it is also essential to consider the properties of the corrosion products, which might be different to those of the original material.

A wide range of investigations have focused on the corrosion and subsequent environmental movement of uranium from nuclear waste. Previous investigations, including laboratory and field studies, have established that natural uraninites (the main form of uranium in ores) and their corrosion products can be used to study the corrosion of uranium compounds in spent nuclear fuel. However, to date it has not been established whether these studies can also be used to describe the corrosion and subsequent environmental movement of the forms of DU and DU-Ti alloys released into the environment during a military conflict.

After their deposition in the soil, the movement in the environment of uranium from DU dusts or intact fragments depends on their rate of corrosion and the rate of dissolution of the corrosion products. The

corrosion and dissolution rates of DU dusts depend upon their chemical composition and size distribution. Uranium oxides constitute the main component of dusts produced from DU during impacts or fires, although such dusts can also contain a mixture of major or trace impurities such as iron, silicon and titanium. These impurities are not present in uranium dusts in the nuclear industry, so studies of the corrosion and dissolution of dusts from the nuclear industry might not necessarily be relevant to DU dusts.

In penetrators, DU is alloyed with a small amount of titanium, which can make its corrosion properties significantly different from those of pure uranium metal. Alloying with titanium reduces corrosion and oxidation, retarding the release of soluble DU into the environment.

Much of our knowledge of the environmental behaviour of DU introduced into the environment comes from studies at sites where DU munitions were tested. For example, a series of experiments and geochemical modelling were used to determine corrosion rates, solubility and sorption (a generic term describing the chemical and physical binding of DU to soil components) of DU in soil at the Aberdeen Proving Ground in Maryland and the Yuma Proving Ground in Arizona. Results from these studies, and from studies performed in the UK at Kirkcudbright, indicate that corrosion rates are highly variable and that under conditions that favour corrosion a 1 cm diameter by 15 cm long penetrator (eg about the same as that in a 30 mm round) would release approximately 90 g of DU per year. For a larger projectile, such as a 120 mm round (3 cm by 32 cm penetrator), this equates to a release of approximately 500 g of DU per year. Based on these corrosion rates, the penetrators will only remain as metallic DU for between five and ten years. Reaction products from the corrosion of DU can be transported as a solid phase by physical processes such as resuspension or can be dissolved in soil water that might become, depending upon local hydrological and environmental conditions, transported into plants, surface waters or groundwaters. During the latter process the migration of dissolved DU is controlled by its solubility under local chemical conditions within the soil water and its sorption onto the immobile soil matrix (both of which could vary significantly over a scale of centimetres). Hence, corrosion rates, the solubility of the corrosion products and the degree of movement of DU in the environment will vary between locations and environments.

2.5 Environmental pathways

Natural uranium and DU differ only in the proportions of the different uranium isotopes and would therefore be expected to behave similarly in the environment. However, when introduced into the environment, DU is present in significantly different chemical and mineralogical forms to those encountered in natural systems in which much of the easily leached or 'labile' natural uranium has already been removed. Consequently, the release of DU into the environment by military conflict can have a far greater impact on the concentration of labile uranium in soil and water than would be expected from its concentration relative to that of natural uranium.

Differences in chemical form between DU and natural uranium, and uranium used within the nuclear industry, also limit the applicability to DU of models and scenarios developed for predicting the behaviour of uranium from nuclear waste. For example, studies of nuclear waste disposal usually focus on transport processes that occur at depths of greater than 100 m below the earth's surface (compared with less than 10 m in the case of DU), and on forms of uranium that are chemically and mineralogically distinct from those likely to be introduced during the use of DU in a military conflict.

The environmental behaviour of uranium is strongly affected by many environmental variables, such as soil composition and chemistry, the level of the water table, the amount of resuspension into the air, climate and agricultural practices. For example, the parameters describing sorption of uranium by different soils vary by a factor of up to one million, even amongst broadly similar soil types. Whilst some authors have suggested that the use of DU munitions is unlikely to add significantly to environmental baseline levels of uranium in soils, it is important to consider that uranium derived from the fragmentation or corrosion of munitions might be more bioavailable, and possibly more mobile in the environment, than the residual uranium naturally present in weathered soils. Such differences have been demonstrated during investigations of soils contaminated by uranium from the Fernald site and at military firing ranges. Also, the relative importance of any additional environmental uranium depends on the depth at which the material is introduced and then how much it is moved into the upper soil layers as a result of agricultural practices.

For example, if 20% of the DU from the impact of a large calibre (4.85 kg) penetrator is converted into dust, as was assumed in the worst-case scenario in Part I of the report, and is evenly dispersed over a radius of 10 m to a depth of 10 cm, it would produce a uranium concentration in the soil of approximately 17 mg per kg. This value is above that observed in most natural soils (typically between 0.5 and 10 mg per kg). However, if a similar release of uranium was restricted to the upper 1 cm or less of soil, as might be expected from the deposition of DU dust on uniform soils of a high clay content, then the resultant concentration, assuming even airborne dispersal, would be in excess of 170 mg per kg. The restriction of elevated concentrations to the

top 1 cm of soil is likely to reduce transfer to most crop plants and to increase intakes by inhalation of DU from resuspension of soil, and from ingestion of soil by grazing animals or by children.

2.6 Airborne transport of DU

Most studies undertaken on proving grounds, or in post-conflict situations, suggest that atmospheric transport of DU occurs over relatively short distances (tens of metres) following the impact of armour-piercing DU projectiles. Longer range transport of airborne particles (tens of kilometres) containing uranium with a natural isotopic signature have, however, been observed in at least one study of airborne uranium concentrations associated with the Kosovo conflict (Kerekes et al 2001). The observation that this increase in uranium concentration (with a natural isotopic signature) could be associated with large amounts of surface dusts introduced into the atmosphere by bombing with conventional high explosive weapons, suggests that the mass of natural uranium introduced into the atmosphere from bombing might well mask any changes in the isotopic signature that would be associated with the release of DU.

Removal of DU particles from the near surface environment (where they can be resuspended) is likely to be relatively rapid, given the apparent corrosion rates. However, data collected in post-conflict assessments (eg UNEP 2001), and studies at proving grounds, suggest that particulate material can still remain on or near the surface after two years.

2.7 Uranium movement in soil

Although the weathering rate of both DU oxides and metallic DU is low, it is still a relatively rapid process compared with that of uranium in many natural soil minerals. However, as for natural uranium, the mobility of weathered DU in the soil profile is dependent upon the affinity of the soil for uranium and the properties of the soil, such as its acidity or alkalinity (pH) and water content. Thus, where soil strongly binds uranium - typically soils high in organic matter have a high affinity for binding uranium - its release into soil water, and movement into groundwater, should be minimal. Correspondingly, mobility is likely to be greater in soils that bind uranium less strongly, which includes those soils in semi-arid environments where neutral to alkaline soil pH is combined with a low organic carbon content. Although the potential mobility of DU should be greater in such semi-arid chalky soils, in practice the lack of water, due to low rainfall and high rates of evaporation, means that migration into deeper soil horizons and groundwater will be reduced.

In environments where uranium is mobile, both point sources of DU, such as intact penetrators or fragments, and diffuse sources, such as DU deposited from aerosols, will gradually disperse throughout the soil. Although this reduces contamination from DU in soil, the enhanced mobility implies that the level of contamination in groundwater might be increased. Similarly, such dispersal of DU might significantly decrease the cost-effectiveness and the technical feasibility of clean-up.

2.8 Migration of uranium into surface and groundwater

The primary factors affecting the potential for DU to contaminate surface and/or groundwater resources, assuming that the uranium is mobile, are the proximity of the contamination to the water source (in the case of surface water) and the water table. For example, groundwater resources associated with river gravels could be particularly vulnerable due to their proximity to the surface. In contrast, the vulnerability of a deeper, possibly confined, underground body of water will be inherently lower. Secondary factors influencing the vulnerability of surface and groundwater to contamination resulting from the use of DU munitions include the chemistry of the water and its local geological environment. These are discussed above within the context of uranium mobility in soils. It is generally considered that uranium mobility in deeper geological environments is much greater than that in soils (provided that such waters are sufficiently oxidising), due to the generally low organic carbon content of rocks and sediments in which aquifers typically occur. A typical deeper geological environment would be an unsaturated zone, which is a region typically lying between soil and an aquifer in which voids are not saturated with water and underlying aquifers.

Whilst the majority of DU might be transported in solution DU particles or fragments might also transport DU into surface waters, reservoirs or groundwater. Transport via such mechanisms has been observed during studies of DU dispersal in weapons proving grounds and test areas.

Perhaps the worst-case scenario with respect to groundwater contamination is that of a DU round penetrating the soil and lodging in a shallow groundwater system (such as an alluvial aquifer). This scenario might directly release uranium into a local water supply, such as a well, as the soil will not be able to act as a 'filter' to prevent any of the uranium entering the aquifer. However, unless the penetrator is directly lodged in a well, even with rapid dissolution such contamination might not be expected to result in a measurable increase in uranium concentration at the

point of use until five to ten years have passed, even assuming reasonably conservative hydrogeological parameters. The best-case scenario with respect to groundwater or surface water is that the penetrator directly enters a highly sorbing medium such as soil with a high organic carbon content, or that it impacts in a clay-rich environment which is effectively impermeable to water, thereby preventing water flow and the migration of dissolved or particulate DU.

2.9 Uranium uptake by micro-organisms, plants, animals and humans

2.9.1 Micro-organisms

The concentration, behaviour and toxicity of DU to micro-organisms are important because: (a) these single-cell organisms lie at the base of many food chains; and (b) they play an important role in influencing the concentration and composition of organic matter in soil, which has been demonstrated to control the mobility and potential bioavailability of uranium in soils.

Reviewed studies indicate a wide range of toxic and cumulative responses in micro-organisms exposed to elevated concentrations of uranium (and hence also DU). Toxicity has been attributed to chemical rather than radiological effects and in comparative studies the levels of observed toxicity were significantly greater than those associated with nickel or copper. Effects of uranium toxicity on soil respiration (reflective of a wide range of soil-associated micro-organisms) were only observed at uranium concentrations exceeding 500 mg per kg. This suggests that such effects are only likely in the immediate vicinity of corroding projectiles or penetrator strikes where concentrations of uranium might exceed this value.

2.9.2 Plants

Most plants take up their nutrients (and contaminants such as uranium) mainly via the roots from the soil solution, although absorption through leaves also occurs. The extent to which uranium or DU is bound to soil components, and the strength of that binding, affects the amount of soluble soil uranium available for uptake into plants. Therefore, the factors influencing uranium mobility in soil are also likely to exert a strong influence on the extent of plant contamination. The uptake of uranium by plants, although low compared with mobile radioactive elements such as caesium and strontium, is higher than that of plutonium and americium. The soluble forms of uranium seem to be readily absorbed by plants; however, in many soils natural uranium has a low solubility and can be unevenly distributed. In general, uranium concentrations in plants decline in the order: roots greater than shoots greater than fruits and seeds

However, atmospherically deposited particles including resuspended soil might significantly increase the concentration of uranium on foliage and unwashed fruits and seeds. The potential for contamination of plants is likely to be very variable due to the presence of highly localised contamination hotspots in soils associated with individual penetrator sites.

Concentration ratios that describe the relative concentration of uranium in plants compared with that in soil have been determined for various sources of uranium (eg mine wastes, tailings and nuclear fuel processing wastes). However, detailed investigations have not yet been reported that study DU-Ti alloys and their corrosion products. Although there are extensive compilations of data, the suggested concentration ratios vary by four orders of magnitude for the same crop on different soils and with different sources of uranium. This wide variation severely inhibits the applicability of generic models that incorporate uranium uptake into plants, and highlights the need for further studies with well-defined source terms and soil compositions.

Studies investigating the toxicity of uranium to plants have produced contradictory findings. For example, indications of toxicity have been observed in grains and other plants at uranium concentrations exceeding 300 mg per kg (soil) or 1 mg per litre (irrigation water). However, a stimulatory effect on growth has been observed in some grasses exposed to elevated concentrations of uranium in soil at broadly similar concentrations. It is therefore impossible to predict the likely impact of DU on plants from a generic perspective without a detailed knowledge of site-specific data relating to the abundance of different species of plants.

2.9.3 Animals

Exposure of animals to DU occurs through pathways broadly similar to those observed in humans, although physiological differences might influence key parameters defining uptake (eg the proportion absorbed from the gut into the blood). The relative importance of each of these exposure routes depends on the physical and chemical nature of the uranium to which individual animals might be exposed. Exposure to naturally occurring uranium can occur via consumption of herbage but in many systems is likely to be dominated by inhalation and ingestion of dusts and soil (either directly or through the ingestion of soil or dusts adhered to the foliage of plants) and drinking water. Exposure to DU is likely to be highly variable due to both differences in animal behaviour and diet, and the highly localised nature of the contamination of soils and food plants.

The extent of systemic absorption via the inhalation pathway in animals depends on the size and chemical form of the inhaled uranium, which influence the

degree to which uranium penetrates the lungs and the rate at which it dissolves in the lung. Uptake of uranium from the gut to the blood is low and, as in humans, most ingested uranium is excreted in faeces, where it might be directly reingested or recycled via the soil into forage. However, although uptake of uranium through the gut is low it is still higher than that of, for example, thorium and plutonium. Recommended gut uptake factors for ruminants are around five times higher than for monogastrics (eg humans). Once taken up the biodistribution of uranium in animals broadly follows that observed in humans (Royal Society 2001) and, compared with other body tissues, high concentrations have been reported in kidney, bone and liver.

Many laboratory-based studies have been undertaken using animals as a proxy to study the potential toxic effects of uranium on human populations (eg ATSDR 1999; WHO 2001). A wide range of toxic endpoints (eg kidney function or morphology, reproductive effects, lung function, etc) were observed in these studies, particularly at high doses (see Chapter 1 and Appendix 1 of this report). Far fewer studies have been performed to assess potential toxicity to domestic animals in the field, although one study of exposure of cattle to uranium at levels similar to those that might result from the use of DU munitions indicated an initial decrease in general health and milk yield followed by an almost complete recovery. Other studies performed at proving grounds in the USA have not indicated substantive levels of toxicity amongst components of natural ecosystems associated with these environments.

There are very few data quantifying the uptake and toxicity of uranium and DU in domestic animal species. It is therefore difficult without the collection of primary experimental data to estimate the potential impacts of the introduction of large amounts of DU into a rural environment. Due to the low uptake of uranium by plants, adherent soil on plants that are ingested by animals might constitute a major source of uranium intake. No data are available on the bioavailability of soil-associated uranium or DU for gut uptake.

2.9.4 Humans

Environmental exposure of humans to DU can occur through three principal pathways: inhalation, ingestion and dermal absorption (eg ATSDR 1999; WHO 2001). As has been discussed in the case of animals, the relative importance of each of these exposure routes depends on the physical and chemical nature of the uranium to which the individual might be exposed. Human exposure to naturally occurring uranium can occur via consumption of a wide range of foodstuffs, all of which contain uranium to some extent, but in many situations is likely to be dominated by inhalation and ingestion of dusts and soil (either directly, or through the ingestion of soil or dusts adhered to the foliage of plants) and drinking water. However, the dominant pathways in the case of DU are dependent upon the nature of the contaminative event and the time elapsed between the release of DU into the environment and exposure. For example, during a conflict the exposure of those in the immediate vicinity of penetrator strikes will be dominated by inhalation (Royal Society 2001), whilst exposure to those living in the vicinity of a combat zone 50 years later might be dominated by ingestion, as the uranium contamination from DU particles and from penetrators has become more evenly dispersed amongst soil, plants and drinking water.

Of the many potential intake pathways associated with ingestion, exposure to uranium or DU in drinking water, milk and soil are considered to be the most important pathways. Intakes by ingestion from soil might be particularly significant in young children and infants. Unsurprisingly, in cultures where the deliberate ingestion of soil is practised (geophagy), soil ingestion represents a dominant pathway even when the low bioavailability of uranium in soil is taken into account. This is because concentrations of uranium in soil are often 10,000 times greater than those in drinking water. Where exposures are limited to accidental or everyday exposures to soils and dusts (eg finger to mouth contact) these form a less important pathway.

In humans the extent of systemic absorption via the inhalation pathway depends on the size and chemical form of the inhaled uranium particles, which influence the degree to which uranium enters the lungs and the rate at which it dissolves in the lung (see Appendix 1 and Annexe A of Part I). Uptake of uranium from the gut to the blood is low and, as in animals, most ingested uranium (about 98% in humans) is excreted in faeces, where it might be recycled via the soil into food or drinking water.

The toxic effects of uranium, and more specifically DU, have been discussed in the first part of the report (Royal Society 2001) and in Chapter 1 and Appendix 1 of this part of the report. Exposures during a military conflict have focused principally on effects associated with acute intakes, and particularly with the large inhalation intakes that might occur immediately following penetrator strikes. Environmental exposures in the years after a conflict are likely to be much lower because of the dispersion of DU throughout the natural environment. However, although these environmental exposures will typically be relatively low, they differ from those that occur on the battlefield as they will be chronic, and thus they require further consideration. Effects on kidney function are the most likely consequences of chronic exposures to elevated levels of uranium, with progressively higher exposures resulting in increasing risks to the kidney and the possibility of radiologically associated risks. However, there are few well-controlled studies of the health effects of chronic long-term exposure of humans to elevated levels of uranium (Royal Society 2001; Chapter 1 and Appendix 1).

Estimates have been made of the amounts of DU that could be inhaled from DU particles resuspended from soil over the years that follow a military conflict and of the subsequent risks to human health (Annexe F). These estimates are clearly subject to considerable uncertainties in the absence of reliable measures of levels of DU particles in the air following a conflict, but they do suggest that the increased risk of lung cancer, or of other cancers is low, and that inhalation is also unlikely to result in any significant effects on the kidney (Chapter 1).

Even using worst-case assumptions, which would only be expected to apply to a few individuals, the estimated lifetime increased risk of fatal lung cancer from environmental inhalation intakes is about six per 100,000, and the central estimate is about six per 10 million. Risks of other cancers (including leukaemia) are at least 100-fold lower than the risks of lung cancer.

Radiation exposure from the inhalation of DU particles is greatest to the lungs and the associated lymph nodes. The possibility that the risks of leukaemia from alpha-particle irradiation of the lung-associated lymph nodes could be greater than those predicted by ICRP models was discussed in Part I of the report. Even if the leukaemia risks from inhaled DU particles are 100-fold greater than those calculated by the ICRP models, the central estimate of risk is still only about three per 10 million.

Intakes of uranium by ingestion from contaminated food and water, or by ingestion of soil, will be highly variable and are very difficult to estimate. There have been several recent studies in Kosovo, which indicate that elevated levels of uranium are not widespread. There are very few published data for Iraq, and attempts to estimate ingestion intakes, and resulting risks, have not been made, although they could be made for specific locations as data become available through continued environmental monitoring. In some situations, such as the ingestion of soil by infants, both chemical and radiological dose limits could be exceeded, although the actual intakes will be related to the frequency of occurrence of these events and the proportion of events in which contaminated soil rather than uncontaminated soil is ingested (Annexe C).

2.10 Case studies

The most extensively researched releases of DU into the environment have occurred at firing ranges or proving grounds. For example, studies of the distribution of DU under various climatic and environmental conditions have been performed at Yuma, Aberdeen and Jefferson in the USA (Ebinger et al 1996; Ebinger and Oxenburg 1997) and at Kirkcudbright and Eskmeals in the UK (MOD 1995) for over ten years. These studies have utilised many techniques, from relatively simply temporal and spatial environmental monitoring against given target levels or threshold levels (often related to radiological rather than chemical toxicity), to more complex studies involving the use of environmental transfer models and the sampling of animals and plants to determine the presence of harm.

At the Jefferson Proving Ground in the USA the results of modelling concluded that no risk to humans occurred from occasional use of the site, the largest exposure to DU being from contaminated dust. Whilst farming scenarios showed some risk of exposure due to inhalation of contaminated dust, by far the largest exposure resulted from the use of contaminated groundwater as drinking water, either by livestock or by humans. The overall conclusions of the modelling exercises were that subsistence farming presented a greater risk of DU exposure than did occasional use. Projections of exposure over the next 1000 years at these sites (Ebinger et al 1996; Ebinger and Oxenburg 1997) indicated a gradual decline of the importance of contaminated dust, and a gradual increase in groundwater contamination over the next 100 years, before reaching a steady concentration between 100 and 1000 years. Obviously such rates are extremely dependent on the exact mineralogy, local soil type and water conditions. The calculated level of risk was extremely sensitive to the solubility of the uranium and it was recommended by the authors that this parameter must not be overlooked when assessing potential risks associated with exposures to uranium or DU from the environment.

Studies performed at proving grounds in the USA have not indicated substantial levels of toxicity amongst components of natural ecosystems associated with these environments. In the UK, monitoring at Kirkcudbright and Eskmeals has not indicated significant changes in the marine environment. In the terrestrial environment, levels of uranium up to several hundred mg per kg of soil have been identified over relatively small areas. These local 'hotspots' have been attributed to material released during firing or when penetrators have veered off target and hit soil or rocks rather than passing through the target and into the sea (MOD 1995).

Studies of potential exposures at military proving or testing grounds provide valuable data, but the amounts of DU used, and the nature of DU munitions use, is often very different from those during an actual conflict. Whilst the relative importance of routes of exposure will probably remain broadly similar, these differences make it difficult to extrapolate the potential exposures and environmental effects from studies at proving grounds to those following a military conflict.

Few studies of the environmental impact and distribution of DU have been reported following the Gulf War, but a relatively large number have been undertaken since the Kosovo conflict (eg MOD 2001; UNEP 2001 and a variety of unpublished studies, including those of Dr C Busby of the Low Level Radiation Campaign and Serbian workers). A striking observation from the environmental assessments in Kosovo is the very low proportion of penetrators recovered in Kosovo (around 10 to 20%). This is consistent with most of the munitions becoming buried in the ground rather than hitting hard targets and producing particulate oxidation products, and the exclusive deployment of 30 mm DU munitions in strafing attacks from A10 aircraft where few penetrators hit their target.

All studies agree that local contamination with DU can be measured up to 10 m from a penetrator strike. However, elevated uranium levels (ie above those of average soils) were generally restricted to less than 1m, and more typically less than 0.2 m, from the actual strike site. Given the variability of potential impacts from a strafing attack of about 250 rounds, covering an area of 200 m by 50 m, a high degree of variation would be expected in the energy dissipated on impact, and thus the percentage of DU oxides produced, depending on the terrain (sandy soil, soft or hard rocks, etc). Absolute uranium concentrations at impact sites varied from a few mg per kg of soil to in excess of 15 g per kg, a level at which significant local effects might be observed in microbiota, plants and animals (see earlier). These areas of local contamination have been highlighted as they could lead to elevated human (or animal) exposure via ingestion, or inhalation, if for example an infant was to play in the immediate vicinity of such a strike. These potential exposures around penetrator impact sites probably represent the only case where acute exposures that are similar in magnitude to those that occur during military conflicts are likely.

To date no studies have observed the presence of DU contamination in drinking water (private wells in the vicinity of strike sites), milk or vegetables. This is not surprising as the timescale of migration and mixing of DU in the soil, and thence migration into groundwater and crops, is likely to be in the order of tens or hundreds of years, and is consistent with the view that a relatively small proportion of the total DU from deployed munitions is converted into DU oxides, which would be expected to have resulted in faster mixing and incorporation into the food chain. However, the presence of the bulk of the DU from deployed munitions as intact penetrators or penetrator fragments that will slowly release uranium into the environment emphasises the need for continued environmental monitoring of water and food supplies over many decades.

2.11 Conclusions and knowledge gaps

Large amounts of DU are introduced into the environment during military conflicts where DU munitions are deployed. Initially this results in exposure of the local inhabitants to DU by inhalation of deposited particles of DU oxides that have been resuspended into the air from soil. Contamination of soil and plants by DU particles will also result in contamination of food and surface waters, and contaminated soil can be ingested inadvertently by infants and children. In the longer term these particles will be removed from the upper layers of the soil, and the environmental movement of soluble uranium from these particles, and from the corrosion of buried DU penetrators, could lead to contamination of local water supplies.

Levels of environmental exposure, and hence any adverse effects on health, will always be less (in the short term) than that of heavily exposed soldiers on the battlefield but, if considerable environmental contamination occurs, the numbers of individuals exposed to chronically elevated levels of uranium could be large, and the total health effects could potentially be as great in the long-term. However, no substantial DU contamination has been measured in Kosovo, except in the vicinity of penetrator strikes, although the situation in Iraq is much less clear.

Modelling of the amounts of DU resuspended from soil in the years following a conflict indicates that the estimated inhalation intakes will not lead to any increase in the incidence of lung cancer or any other cancers among children or adults. Nor are they likely to lead to any significant effects on kidney function. The accuracy of such modelling is sensitive to the selection and validity of the parameters that are used in the models (eg the intakes of DU), which are highly dependent on local environmental conditions, the amounts of DU munitions that are deployed and the nature of their use (eg large calibre munitions against tanks compared with small calibre munitions in strafing attacks).

There are clearly major uncertainties that limit any evaluation of the environmental consequences of the use of DU munitions and particularly those that arise from ingestion. The intakes from ingestion of soil, or from contaminated food and water, will be highly variable as both the deposition of DU particles and the distribution of buried penetrators will be dependent on the military events that occurred within the area. A major problem is that most DU penetrators used in a conflict are expected to be buried. Thus, very few of the DU penetrators fired in the Gulf War or in Kosovo have been recovered; it is assumed that about 80% penetrated the soil, but their distribution in the soil is largely unknown. There are also few data on the amounts of DU oxides released for the many different types of impacts that can occur (eg soils, rocks,

buildings, as well as military vehicles), and the environmental behaviour of the DU-Ti alloys used in DU rounds, and the derived particles of DU oxides, will differ from that of naturally occurring uranium minerals.

Furthermore, the rate of corrosion of buried DU penetrators will vary considerably depending on local soil conditions, and this variability, together with the unknown distribution of penetrators, the wide variability in the possible rates of environmental movement of uranium, the variability in human behaviour, and variability in the proximity of penetrators to susceptible water sources, makes it difficult to produce any general estimates of intakes or health risks from ingestion of contaminated food or water.

Estimates of the health risks of intakes from ingestion have therefore not been attempted. There are, however, some scenarios where, on a local scale, levels of uranium intakes by ingestion could be elevated and which could be a cause for concern. In particular, hotspots of contamination will occur which could result in substantial intakes for a few individuals, eg a child playing at the site of a penetrator strike, or ingestion of food grown on areas of local contamination, or where a DU round feeds uranium into a local water source. Site-specific modelling even with minimal site-specific data should be an inherently more reliable approach than general modelling approaches to estimate the possible risks in these specific scenarios.

Environmental movement of uranium will be slow (decades) and the absence of any significant contamination in drinking water does not necessarily imply that elevated levels of uranium will not occur in some local supplies in the future. Drinking waters that are derived from small lakes within an area where a conflict occurred, or from shallow groundwater sources, are particularly at risk of contamination. Continued monitoring for contamination is therefore important and needs to continue over several decades.

3 Responses to Part I of the report

3.1 Introduction

After the publication of Part I of the report a public meeting was held to discuss the conclusions that were reached about the radiological risks of the use of DU munitions. A number of issues were raised at this meeting and also in correspondence and meetings with further experts and veterans. One feature of the report that was not well understood was the need to use modelling as a tool for predicting the likely radiological consequences of DU exposure where reliable direct measurements of any adverse health effects (predominantly an increased risk of lung cancer) are unlikely to be available for many years. The importance of modelling is discussed in Section 3.2.

The discussion of the radiological effects of DU in Part I was restricted to the increased risks of cancer. During the public meeting it was suggested that we look at the possibility of radiological effects on the immune system. This is considered in Section 3.3.

The estimates of the increased risks of cancer from the radiological effects of inhaled DU, and of kidney disease from the toxic effects of elevated levels of uranium, are dependent on the intakes of DU in different battlefield scenarios. As discussed in Part I, these are subject to considerable uncertainty, but the central estimate and worst-case values of intakes we used in Part I (and the derived estimates of risk) can be adjusted as new data become available. Evidence about intakes during the Gulf War has been taken from Dr Doug Rokke who was part of a US army unit involved in the damage assessment and clean-up of vehicles struck by DU munitions. It was stressed by the veterans groups and their advisors that Dr Rokke had first-hand evidence of the extent of DU contamination following the Gulf War that was crucial to our study. We therefore talked with him at length by videolink, corresponded extensively and received a number of documents from him.

The importance of evidence collected by Dr Asaf Durakovic and Dr Pat Horan on uranium isotopes in the urine of a group of Gulf War veterans was also stressed by the veterans groups and their advisors. Dr Durakovic gave evidence to the working group and these studies of urinary uranium levels and the evidence obtained from Dr Rokke are discussed in Section 3.4.

3.2 Modelling

In Part I assessments were made of the intakes of DU which might occur on a battlefield in which DU weapons are used, of the resulting radiation doses to various body tissues and organs, and of the excess risks of various cancers resulting from the radiation. In Part II assessments have been made of the concentrations of uranium in body tissues, particularly in the kidneys, resulting from intakes of uranium, and of the effects of these concentrations on kidney function. To make these assessments, 'models' were used extensively to calculate the various quantities, such as the amount of uranium that might be inhaled, and how much ends up in the different tissues at any time after the exposure.

Models make use of scientifically based, quantitative, descriptions, which include known physical, chemical and biological mechanisms as far as possible, and the available experimental information. Models are tested as more information becomes available, and they evolve as their scientific base is improved. Sophisticated and realistic scientific models (not to be confused with simplistic qualitative descriptions) are valuable because (a) they bring together a large amount of established knowledge in a systematic way, (b) they can be used to check the consistency of information from different sources, and hence identify conflicts, (c) they can be used to analyse a range of scenarios in strictly comparable ways, and (d) they allow one to estimate sensitivities to assumptions and to establish crucial gaps in data. They allow one to relate data from widely different types of information, and they can make possible the interpretation and understanding of what is important in complex situations in which there are many inter-related factors.

Models are widely used in both the biological and physical sciences and their applications, in areas ranging from aircraft engine design and ballistics to public health. For example, in studies of infectious disease, models have been used to predict the course of epidemics and are particularly useful as they allow the relative efficacy (or cost-effectiveness) of different possible control measures to be predicted. Models evolve and their accuracy at predicting events improves as new experimental data are obtained. They provide the only valid approach to obtaining a scientifically rigorous assessment of the course of future events where experimental data relating to such events are not yet available. For example, in the physical sciences, models of increasing sophistication and accuracy have been used for hundreds of years to predict the movements of planets and other heavenly bodies, allowing the precise timing of eclipses and the trajectories of comets and asteroids to be accurately determined.

Most scientists accept that the modelling approach is appropriate for estimating the risks of exposure to DU, given the following:

- There is no direct evidence from human (epidemiological) studies that can relate cancer risk to exposure to DU aerosols such as those likely to occur on the battlefield.

- There is, however, a considerable amount of information available on the way uranium behaves after it enters the body. There is also convincing evidence from both human and animal studies that irradiation of at least some body tissues (including lung, bone and bone marrow) does cause an increased risk of cancer which increases with radiation dose, at least at moderate to high doses (above 100 millisieverts).
- There are animal data, and some human data, on levels of uranium that are toxic to the kidney, but direct measurements of concentrations of uranium in the human kidney are not feasible, and the levels can only be estimated from measurements of uranium concentrations in urine or from the likely intakes.

Modelling provides limits to the likely range of possible adverse events that can be narrowed as additional data become available. Thus, in the case of DU munitions, better measurements of the amounts of DU released into the environment during an impact with a target, and of the size distribution and the solubility in lung fluid of the resulting DU particles, are required to provide better estimates of the risks to health. The models used by the International Commission on Radiological Protection (ICRP) are rigorous and scientific and contrast sharply with often anecdotal assessments of the health of soldiers and of inhabitants of areas where DU munitions were deployed. However, modelling is not a substitute for directly measuring the health effects of exposures to DU, which requires very carefully designed long-term epidemiological studies of exposed soldiers, but it provides estimates of the likely outcomes given the available information.

3.3 Immunological effects from exposure to DU

At the public meeting to discuss Part I it was suggested that we should examine whether radiation from internalised DU might have adverse effects on the immune system. Although Part II of the report focuses on the chemical toxicity of uranium, the possibility of radiological effects on the immune system is considered here.

3.3.1 Immune effects following the atomic bombs in Japan and the accident at Chernobyl

Effects of acute high exposures to direct whole-body irradiation on the immune system have been studied in the survivors of the atomic bombs in Japan. These studies initially showed no significant dose-related effects using a wide range of immunological tests (Finch 1979; Akiyama et al 1991), although subsequent studies carried out 30-40 years after the events showed effects on the numbers and function of some cells of the immune system (T cells), which have become more clear 50 years after the bombings (Kusunoki et al 2001). However, these effects, resulting from whole-body irradiation (mainly gamma-radiation), may have little relevance to the situation with DU where the main exposure is radiation of the lung and associated lymph nodes from alpha-particles following inhalation of aerosols produced after the impacts of DU penetrators with tanks.

There are also some minor effects on immune function in workers involved in cleaning up after the Chernobyl accident in 1986, but these workers received direct irradiation, as well as inhalation of particles containing radionuclides such as ^{90}Sr, ^{134}Cs, ^{137}Cs, ^{239}Pu and ^{240}Pu. The studies of Chernobyl workers have been reviewed recently by UNSCEAR (2000), who concluded that no immunological defects could be associated with ionising radiation caused by the Chernobyl accident. According to UNSCEAR, direct effects on the immune system would not be expected at the doses of radiation received by the Chernobyl workers and they have suggested that psychological stress could have caused the fluctuations in some immunological parameters in different groups of exposed Chernobyl workers.

3.3.2 Immune effects from discharges of highly radioactive waste from the Mayak nuclear plant

In the 1950s several hundred workers in the Mayak nuclear plant in the Southern Urals, and nearly 1,000 residents in villages along the Techa River, into which large amounts of high-level radioactive waste were discharged, became ill and were diagnosed as suffering from a chronic radiation syndrome (AFRRI 1994, 1998). The radiation doses received by these individuals are considered to be the greatest known chronic environmental exposures of a human population. Protracted doses to the red bone marrow of combined external gamma-rays and internal exposures, mainly from ^{90}Sr (strontium-90), had a median accumulated value over 25 years of around 0.25 gray (Gy) and a maximum of about 4 Gy. The highest levels were found in the first years of exposure, and 80-90% of all doses due to internal exposure were accumulated in the first ten years. The syndrome was characterised by neuroregulatory and cardiovascular disorders, moderate reductions in white blood cells and, in severe cases, a weakened general immunity with infections or septic complications. Changes in immune status, and increased infections, were apparent over a number of years in this population and have been attributed largely to the intakes of ^{90}Sr, a highly radioactive bone-seeking radionuclide, which result in many years of radiation exposure of the red bone marrow, one of the central organs supporting the immune system (Akleyev et al 1999). During the first two to four years after the onset of chronic exposure of the Techa riverside inhabitants, changes observed in the peripheral blood were manifested by leukopenia (mostly due to reduced neutrophil counts) and thrombocytopenia, at equivalent dose rates to the red

bone marrow of 300-500 millisieverts (mSv) and higher per year. The threshold dose causing reduced resistance to infections (based on tests for skin autoflora) was estimated as 300-400 mSv per year to the red bone marrow in these conditions of chronic exposure (Akleyev et al 1999). As the years progressed (43-48 years after the beginning of the exposure) the production of blood cells and immunity was normal among most of the exposed subjects. However, some of the individuals were still noted to show an increased frequency of chromosomal aberrations (both stable and unstable types) and of mutant T-lymphocytes in the peripheral blood (Akleyev et al 1999).

3.3.3 Immune effects in animals following inhalation of alpha-emitting particles

In the Chernobyl workers, and the exposed Mayak and Techa River populations, it is difficult to untangle the roles of external radiation, internal radiation from highly radioactive bone-seeking radionuclides, and psychological stresses in the alterations of immune function. Animal studies circumvent these problems and allow the effects of the intakes of known amounts of a single radionuclide to be related to effects on immune function. The most relevant studies for populations exposed to DU aerosols are the experiments where the immune status of dogs has been examined following inhalation of alpha-emitting radioactive particles (typically $^{239}PuO_2$; plutonium oxide). In these studies effects on the levels of white blood cells (lymphocytes and neutrophils) have been identified, as well as atrophy of lung-associated lymph nodes due to the deposition of the particles in these lymph nodes and irradiation of resident and trafficking cells (Davila et al 1992; Weller et al 1995; Muggenberg et al 1996, 1999; Park et al 1997). However, these effects have not been associated with any obvious deficiency in immune function or any increased incidence of infections, and they occurred at very high radiation doses. High doses were achieved by using $^{239}PuO_2$, which is highly radioactive, and they could not easily be achieved following inhalation of a weakly radioactive material such as DU. For example, most of the observed effects on particular components of the immune system occurred at radiation doses that for a human that would require the retention of at least 20 g of DU particles in the lungs. Assuming retention of 20% of the intake in the lungs, this would correspond to the inhalation of more than 100 g of DU oxides.

3.3.4 Immune effects from exposures to DU

Some killing of lymphocytes by alpha-particles from retained particles of DU will occur as the lymphocytes pass through the lung-associated lymph nodes of soldiers exposed to aerosols of DU, but these are unlikely to lead to any significant reduction in the ability of the body to combat infection. Reductions in immunity would require continuous effects on the mature lymphocytes or on the precursor cells in the lymphohaemopoietic organs, including the red bone marrow.

For most battlefield scenarios the estimated doses to the red bone marrow are much less than the normal doses to this tissue from natural background radiation. The highest dose to the red bone marrow would be from the worst-case Level I scenario, where it can be calculated that inhalation of 5000 mg DU (the intake used for the worst-case Level I exposure scenario) would give an estimated equivalent dose to the red bone marrow of about 12 mSv during the first year, and total doses of 26 mSv after 5 years and 55 mSv after 50 years, using the worst-case estimate of radiation dose per unit intake for the red bone marrow based on the chemical toxicity worst-case (highest solubility of DU).

Using other worst-case assumptions the doses to the red bone marrow are less. Thus, for the worst-case assumptions that maximise radiation exposure to the lungs (lowest solubility of DU), the estimated total equivalent dose to the red bone marrow from Level I exposure after 50 years would be 13 mSv (see Part I of the report).

The doses averaged over several years from even the worst-case Level I intakes are not very much greater than the doses to the red bone marrow from natural sources (about 1 mSv per year), and are much lower than those demonstrated to cause deficiencies in immune function in humans from chronic irradiation of the red bone marrow (doses above about 300-400 mSv per year; Akleyev et al 1999).

This comparison has been made on the basis of equivalent doses (in mSv) to red bone marrow, which implicitly include the radiation weighting factor of 20 for alpha-particle irradiation, according to the ICRP (1991) prescription. Their choice of that factor, however, was based on considerations of cancer risk not immunological effects, for which an appropriate weighting factor, or relative biological effectiveness, has not been determined. If the immunological effects are primarily the result of cell killing by the radiation, a weighting factor of less than 20 is likely to apply, with correspondingly decreased expected effects.

It is concluded that inhalation of DU on the battlefield is very unlikely to result in significant effects on immune function that would increase susceptibility to infection. Whether there could be slight but clinically insignificant defects in immune functions in soldiers with very high intakes of DU, which could add to similar defects from the other toxic exposures that may have occurred in the Gulf War, to produce an overall health detriment, is more difficult to evaluate.

3.4 Exposure to DU in soldiers cleaning up struck vehicles during the Gulf War

The extent of contamination in struck vehicles and the estimates of intakes of DU used in Part I of our report have been discussed with Dr Doug Rokke, who was part of a unit involved in damage assessment and clean-up of allied and Iraqi tanks during the Gulf War. Dr Rokke was also involved in DU 'burn' tests and 'impact' tests in Nevada during the mid-1990s. Most of our discussions have been concerned with estimates of the intakes of DU that occurred in the Gulf War, and particularly in Dr Rokke's unit, which possibly included the US soldiers most heavily exposed to inhaled or ingested DU in this war.

3.4.1 Intakes for heavily exposed soldiers in the Gulf War

Dr Rokke suggested in his evidence that even our worst-case intakes may in some cases be too low. From his personal experiences during the Gulf War, Dr Rokke considers that US and Iraqi vehicles were typically struck by four or five large calibre DU rounds. However, detailed reports of the 'friendly fire' incidents (OSAGWI 2000) state that only one of the six US tanks involved in these incidents was hit by three DU rounds, another was hit by two rounds and the other four by a single round. Similarly, of the 15 Bradley Fighting Vehicles involved, one was hit by three rounds, six by two rounds and the other eight by a single round. There is a conflict between the report from the Office of the Special Assistant for Gulf War Illnesses (OSAGWI) and the oral evidence provided by Dr Rokke. Furthermore, a battlefield assessment memo, dated 31 March 1991 and co-authored by Dr Rokke, is consistent with OSAGWI and states that most tanks were struck by one or two rounds, and that no tank was struck by more than three rounds, and it therefore contradicts the oral evidence provided to the working group by Dr Rokke. We have nevertheless considered a new worst-case intake assuming a tank was struck by three large calibre DU penetrators (Section 3.4.2).

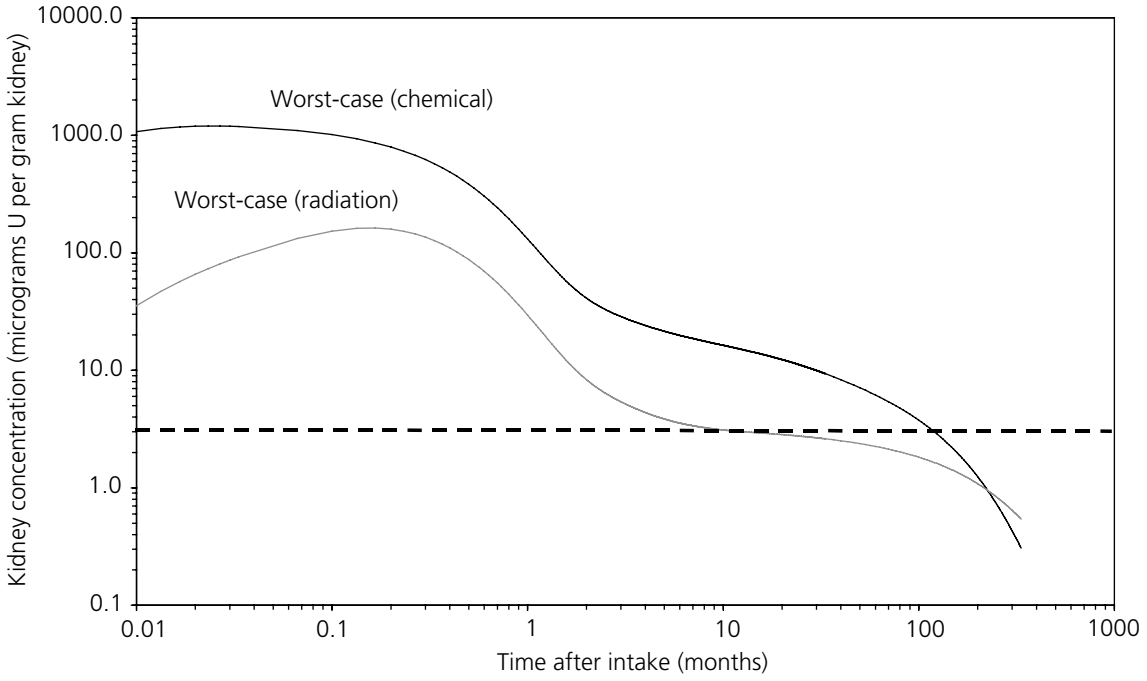
Dr Rokke also suggested that Level II exposures to DU may in some special cases have been greater than those we considered and, for a few soldiers following the Gulf War, were even greater that those occurring in our worst-case Level I scenario (intake of 5 g of DU oxides). According to his evidence, soldiers surviving in tanks would have quickly applied their face mask to help them breathe within the struck vehicle, and in most cases would have been exposed to high DU concentrations for only one or two minutes, rather than the 60 minutes we assumed. Reducing the exposure duration to three minutes would only reduce our worst-case Level I intake to about 3 g of DU, because we assumed that the DU concentration fell rapidly (Part I, Annexe C, table C2). In contrast, he claims that those in his unit were working in or around DU-contaminated vehicles all day, every day, for about three months. Using his estimates, members of the unit worked for six or seven hours inside struck vehicles every day for about three months, resulting in a total exposure time to resuspended DU within vehicles of about 600 hours. We are unable to confirm this estimate, but it compares with our worst-case Level II estimate of 100 hours working within contaminated vehicles (total intake of 2 g DU), and our central estimate of ten hours exposure (total intake of 10 mg).

Dr Rokke's evidence again conflicts with official US military sources, but in this case by a much wider margin. OSAGWI tasked the US Army Center for Health Promotion and Preventive Medicine (USACHPPM) to perform exposure, dose and risk estimates for the 13 exposure categories within Levels I, II and III. A summary is given in OSAGWI (2000), Tab O.

Based on interviews with Level II personnel and analysis of their possible activities, USACHPPM concluded that Level II personnel encountered some or all of the following contaminated vehicles: 16 Abrams tanks (six destroyed by 'friendly fire', three destroyed intentionally, seven involved in fires) and 15 Bradleys (all involved in 'friendly-fire' incidents). They also concluded that one person, exposed to all 31 vehicles, provided a very conservative estimate of the upper limit exposures for Level II personnel. They considered six groups of personnel within Level II and on this basis they assessed intakes in the range 2-8 mg (OSAGWI 2000; table O4), somewhat lower than our Level II central estimate.

Another consideration raised by Dr Rokke is that exposure to DU for a soldier surviving in a tank struck by a DU round (Level I) is predominantly from the impact aerosol and shrapnel, whereas the release of additional DU from unfired rounds in struck tanks may result in more extensive DU contamination in tanks that burn out after DU impacts. The additional contamination from the stored rounds is difficult to estimate but would probably be relatively slight since Iraqi tanks did not carry DU rounds and the additional contamination would only apply for soldiers working on the six US tanks involved in the 'friendly fire' incidents, three of which apparently burnt out (OSAGWI 2000; Tab H). Additionally, there were four tanks damaged in fires at Camp Doha (OSAGWI 2000; Tab I) and four other tank fires (OSAGWI 2000; Tab J). Many of the stored penetrators in these tanks were recovered intact or with minor oxidation damage, although in a few cases (eg tank B23; OSAGWI 2000; Tab J) some or all of the loaded DU rounds appear to have been destroyed in fierce fires. The amount of DU released depends on the intensity and duration of a fire and is believed to be less than that released in impacts with a tank (Part I, Annexes G and H). The size distribution is also different, with much larger particles being produced in fires, resulting in a far smaller proportion of the released DU being in the respirable range (<1% compared to about 50%: Part I, Annexes G and H).

Figure 3.1. Estimated levels of uranium in the kidney following an acute inhalation of 15 g of DU oxides from DU penetrator impacts (Level I). The levels are shown using worst-case (chemical toxicity) and worst-case (radiation) parameters. A concentration of 3 µg uranium per gram kidney is shown by the bold dashed line. For details of the assumed exposure conditions (aerosol size distribution, dissolution characteristics, etc), see Part I, Appendix 1, table 14.



Additionally, from tests in Nevada, Dr Rokke believes that about 2000 g of DU (about 40 % of the mass) is released into a tank struck by a 120 mm DU round. This value appears to come from a single test firing into a tank and was obtained by sweeping up and weighing the debris (assuming it all to be DU) within the previously clean tank. This value is higher than those calculated from most other reported tests of penetrator impacts with armour plate in which, with one exception where there was an estimated 70% release, about 0.1-30% of the DU was released (Part I, Annexe C, table C1). This led us to use a worst-case value of 1000 g (about 20%) of DU released from a 120 mm round. It is not clear that the simple measurement described to us by Dr Rokke is any more reliable than the other estimates and, without new data from the test firing programme that was recently completed in the USA, we see no reason to change our worst-case estimate.

3.4.2 New worst-case intake and health risks for a Level I exposure

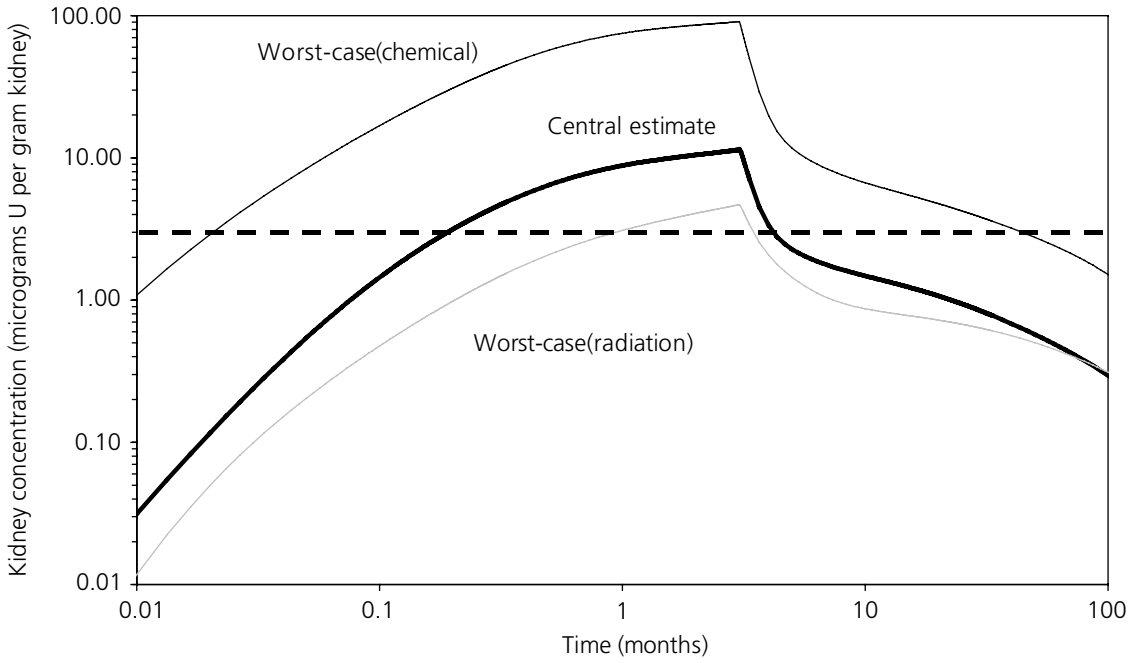
The new worst-case Level I estimate uses the value of 1000 g of released DU per 120 mm penetrator, but with three impacts per tank. If we use our previous assumptions (50% of the released DU is respirable and the survivor is trapped in the tank for one hour) we obtain an inhalation intake that is three times greater than our previous estimate (15 g of DU oxides compared to 5 g). This value assumes that all three penetrators enter the crew compartment, although it seems unlikely that anyone would survive in a tank struck in the crew compartment by three large calibre DU penetrators. It is also very unclear whether it is possible to inhale 15 g of DU oxides in such a short period without choking and it assumes that a soldier inhaled DU for one hour and did not reduce the intake by applying his face mask. If such a very large acute inhalation intake occurred it is predicted to lead to an extremely high worst-case peak kidney uranium concentration (about 1200 µg uranium per gram kidney; figure 3.1), which inevitably would result in acute kidney failure and death without intensive medical care.

The worst-case risk of fatal lung cancer would increase proportionately for the worst-case Level I scenario involving three penetrators, from an estimated excess risk of 6.5 per 100 for one penetrator (Part I of the report) to about 20 per 100 for three penetrators, assuming that the dose response remains linear at these very high lung doses. However, even under worst-case assumptions for radiation (low solubility in the lungs), the peak kidney concentration would be high enough to lead to acute kidney failure (160 µg uranium per gram kidney).

We are not aware that any soldiers involved in 'friendly fire' incidents had acute kidney failure within a few days of exposure, although we have no knowledge of effects in Iraqi soldiers surviving DU impacts, and for this reason, and the other reasons discussed above, such large Level I intakes seem unlikely to have occurred.

Our central estimate of intakes and risks for Level I exposure have not been changed but they could be

Figure 3.2. Predicted kidney concentration following protracted Level II inhalation exposure (130 mg DU oxides per day for 90 days for each case). The levels are shown using central estimate (bold line) and worst-case (chemical toxicity) and worst-case (radiation) parameters. A kidney concentration of 3 µg per gram kidney is shown by the bold dashed line. For details of the assumed exposure conditions (aerosol size distribution, dissolution characteristics, etc), see Part I, Appendix 1, table 15.



scaled up if there was clear evidence that during a military conflict tanks were typically struck by more than one large calibre DU round.

3.4.3 New estimates of risks for soldiers exposed to very protracted Level II intakes

Dr Rokke suggested that greater intakes of DU than we considered previously occurred in some soldiers cleaning up struck vehicles. In Part I, for worst-case Level II intakes, we considered that each tank was struck by two large penetrators. As the written reports state that more than half of the tanks and Bradley Fighting Vehicles involved in the 'friendly fire' incidents were struck by a single DU round, and only two of the 21 vehicles were struck by three rounds, we consider two impacts to still be appropriate for the worst-case average number of large calibre penetrator impacts per vehicle.

If we assume two impacts per vehicle as a worst-case, increasing the maximum exposure time from 100 hours to 600 hours would increase the total worst-case intake from 2 g to about 12 g of DU (about 130 mg of DU inhaled per day for three months). However, this value would almost certainly be too high as some of the vehicles that were entered would have been lightly armoured vehicles, where it is considered that much less DU is released as an aerosol (although this point is disputed by Dr Rokke). However, we have used this figure as a worst-case intake for this group of soldiers since we have not increased the intakes of DU to reflect the possibility that a few tanks were more heavily contaminated than we estimated previously. There would also be some ingestion of DU, but this would only be expected to add a few per cent to the maximum kidney concentrations achieved from inhalation (Part I, Appendix 1, table 27).

Using the worst-case assumptions for chemical toxicity (ie assuming the highest likely value for the solubility of DU oxides) the inhalation intake of 130 mg of DU each day for three months predicts a peak uranium level of about 100 µg per gram kidney (figure 3.2), which would probably result in acute kidney failure. According to Dr Rokke none of his unit suffered obvious kidney problems during the Gulf War, although he stated that kidney problems have subsequently become apparent (see below). This suggests that the kidney uranium levels in this group of soldiers were well below those estimated from the above worst-case intake and the worst-case assumptions about the solubility of the DU oxides.

For these intakes and using the worst-case parameters for radiation, the excess risk of fatal lung cancer is estimated to be about 15 per 100, and this dominates the overall cancer risk. Estimated fatal cancer risks to all other tissues individually are less than 1 per 10,000, with red bone marrow, bone surface and colon being the highest.

Our assessments for members of this unit have provided a worst-case estimate of their health risks which assumes the very large intake of DU (130 mg inhaled

Table 3.1: Predicted maximum concentrations of uranium in the kidney following daily inhalation intakes of 130 mg DU oxides for three months. Values are shown for central estimate, and worst-case (chemical) and worst-case (radiation), parameters.

	Maximum kidney concentration (µg uranium per gram kidney)	Number of months above 3 µg uranium per gram
Central estimate	10	4
Worst-case (chemical)	100	40
Worst-case (radiation)	5	3

per day for 3 months), and the values of other relevant parameters (solubility of the DU oxides, etc) that maximise radiation dose to the lungs (worst-case radiation), or that maximise the level of uranium in the kidney (worst-case chemical toxicity). We have already provided central estimates of risks for a typical soldier working in struck tanks (Level II) but, for Dr Rokke's unit, we can also consider the central estimates of risk for any soldier in the unit who did receive such a very large intake of DU (ie 12 g), by using the central estimates of the solubility of DU and of the other parameters of the biokinetic models.

In this scenario, the maximum level of uranium is predicted to reach about 10 µg per gram kidney and to remain above 3 µg per gram for about four months. This level would be expected to result in significant kidney dysfunction although it is unlikely that it would lead to acute clinical signs of kidney disease. The excess lifetime risk of fatal lung cancer is estimated to be about 3 per 100, and that for fatal cancers of any other tissue to be less than 1 per 10,000 (red bone marrow, bone surface and colon being the highest).

It should be stressed that the increased estimates of Level II inhalation risks for the group of soldiers involved in cleaning up struck vehicles during the Gulf War should not be extrapolated to other soldiers on the battlefield, as the increases are mainly due to the very protracted duration of the high intakes in this particular group suggested by Dr Rokke. We see no reason at present to make any substantial changes to the central estimate intakes, and therefore the risk estimates, that we have reported in Part I for Level I, II or II exposures. The possibility of increased levels of contamination within some struck vehicles could slightly increase the intakes of DU for soldiers briefly entering a struck tank, or for medical personnel handling contaminated casualties, but the effects on cancer risks, or kidney effects, are small since the central estimates of risk from these Level II and III exposures are low.
In any future conflict using DU munitions, large Level II intakes should not occur due to the much greater awareness of possible exposures to DU, and consequent risks to health, and thus the clear need for effective respiratory protection when working in struck vehicles.

3.4.4 Health effects

In his evidence, Dr Rokke claimed that about 20% of the men in his unit have died, mainly from lung cancer, but also from cancer of the pharynx and from lymphoma, and that the others all are sick. These values for morbidity and mortality are anecdotal and we have no way of confirming them.

Members of the unit are claimed to be suffering from a number of similar symptoms, including kidney stones (which Dr Rokke argues come from uranium precipitating in the kidney), renal colic, bone effects, reactive airway disease, rashes, gastrointestinal effects, increased susceptibility to infection and memory loss. These health effects are difficult to evaluate objectively but they suggest a pattern of illness that is different from that expected from uranium exposure. For example, we are not aware of any published reports of gastrointestinal effects or kidney stones from uranium exposures in man or laboratory animals, and decreased immune function would only be expected at very high DU intakes that would almost certainly be lethal due to kidney toxicity.

Interestingly, according to Dr Rokke none of the unit appears to have had acute kidney disease while working in the Persian Gulf, which puts an upper limit on their uranium intakes. Thus, it would appear that no soldiers in the unit had kidney uranium levels that are close to those predicted from the above worst-case intakes and worst-case assumptions about the solubility of DU oxides. However, if soldiers in this unit received intakes approaching those used as a worst-case, the possibility of serious kidney damage is very real using central estimates of DU solubility, and the increased risk of lung cancer would also be substantial.

The possibility of large intakes of DU, and the anecdotal reports of the mortality and morbidity in Dr Rokke's unit, warrant an independent evaluation of the health and mortality in this potentially highly exposed group of Gulf War veterans.

3.4.5 Measurements of DU in the urine of Gulf War veterans

Levels of uranium isotopes in the urine of 27 US, Canadian and British Gulf War veterans have been measured in the laboratory of Dr Pat Horan using Thermal Ionisation Mass Spectrometry (TIMS). The results were presented to the

working group by Dr Asaf Durakovic. The presence of DU in urine can be identified by detecting an increase in the normal ratio of the uranium-238 and uranium-235 isotopes. Natural uranium has a $^{238}U/^{235}U$ ratio of about 138 whereas the DU used in penetrators has a ratio of about 490. Isotope ratios that are significantly greater than 138 imply that a proportion of the uranium present in urine is DU and, the greater the isotope ratio, the higher the proportion of the total uranium that is DU. Determining the total concentration of uranium in urine, and the proportion that is DU, allows the concentration of DU in the urine to be measured.

The data from Dr Horan showed an increased isotope ratio in 13 of the 27 veterans. However, the reported levels of uranium in the urine of most of the veterans was low (similar to that typically found in the general population), and obtaining reliable estimates of uranium isotope ratios in such urine samples using TIMS is challenging, and subject to a number of potential problems. An increased $^{238}U/^{235}U$ ratio can occur for a number of reasons (for example, due to natural exposure to small amounts of DU which in recent years has been increasingly present in the environment). Non-linear effects that lead to higher measured $^{238}U/^{235}U$ ratios at low concentrations have also been observed and isotope measurements involving low uranium concentrations in urine will be prone to artefacts (Thirlwall 2001). Contamination of reagents with tiny amounts of DU from external sources is also a significant problem. These possible artefacts highlight the difficulties of measuring uranium isotopes in urine, which is much more challenging than geological analysis as the concentrations involved are considerably lower.

The ability of a number of laboratories to measure reliably small amounts of DU in urine is being examined by the Ministry of Defence's DU oversight board as an essential prelude to determining whether DU is detectable in urine from veterans of the conflicts in the Persian Gulf and the Balkans.

A serious limitation of the Horan study of Gulf War veterans is that levels of uranium isotopes have not been measured in any control group. This is absolutely essential when trying to measure altered isotope ratios in urine containing very small amounts of uranium. Unless it can be shown that the $^{238}U/^{235}U$ ratios in some of the veterans are significantly greater than those found in the urine of a control group that was not deployed to the Persian Gulf, the results of Horan and colleagues are inconclusive. The presence of DU in urine of these veterans has been widely publicised and checking the validity of these measurements by using an appropriate control group is crucial since, if they are correct, they indicate that DU can still be detected in urine samples taken about 9 years after the Gulf War, which may help to assess the likely intakes of DU received by these veterans. However, if they are incorrect, they will have raised very considerable unwarranted anxiety in those veterans who believe they have 'tested positive' for DU.

Attempts to validate uranium isotope measurements in urine are urgently required so that reliable measures of DU in veterans of the Gulf War can be obtained.

However, detecting the presence of DU in urine, even at such a long time after exposure, does not in itself mean that the intake was large enough to be likely to cause any observable health effects. The average amount of uranium excreted in urine from natural sources is about 0.01 µg per day (Ting et al 1999). Suppose that excretion of 0.005 µg DU per day in urine could be measured reliably in the presence of 0.01µg per day of natural uranium. We can estimate the original inhalation intake that would result in such a level 10 years after the exposure. For inhalation under our assumed central estimate Level II conditions, the ICRP models predict that from inhalation of 1 g DU oxide urinary excretion after 10 years would be about 0.2 µg per day. Excretion of 0.005 µg per day is therefore predicted from an intake of about 25 mg DU oxide. This is somewhat higher than our central estimate for Level II (10 mg), but the associated committed effective doses and maximum kidney concentration are still small. For our central estimate these were 0.5 mSv and 0.05 µg per gram kidney, respectively. For an intake of 25 mg the dose would be predicted to be about 1 mSv, similar to that from natural background each year, and the maximum kidney concentration about 0.1 µg per gram kidney, which might possibly produce a transient change in kidney function that could be detected using sensitive biochemical tests on urine samples, but would be very unlikely to produce any clinical sign of kidney damage.

This back-calculation procedure is subject to very considerable uncertainties, particularly in the fraction of the intake excreted in urine each day at such a long time after the intake, but the calculation indicates that reliable measures of DU in urine could still be useful in assessing the magnitude of intakes of DU during the Gulf War.

3.4.6 Chromosome aberrations in lymphocytes of Gulf War veterans

The presence of increased numbers of chromosome aberrations is well established in individuals who have had sufficiently large exposure to ionising radiation (or some other toxic exposures). Measurement of aberration frequencies in peripheral blood lymphocytes has become a standard biodosimetric technique to estimate retrospectively the doses received in radiation accidents (IAEA 1986; Tucker et al 1993; Finnon et al 1995). Radiation-related increases in frequencies of aberrations have been detected in a wide variety of populations, including nuclear workers, patients after radiation therapy or diagnostic medical exposures, from accidents and from high natural background radiation, including both internal and external radiation sources (IARC 2000; 2001).

Increased frequencies of chromosome aberrations in lymphocytes have been observed in underground uranium miners (Brandom et al 1978; Sram et al 1993; Popp et al 2000). These detectable effects in blood cells have been attributed mostly to exposure to radioactive radon gas and its short-lived decay products. Characteristic aberrations have also been described in non-malignant bronchial epithelial cells from lung cancer patients and cancer-free former uranium miners and smokers (Crowell et al 1996; Neft et al 1998). An increased frequency of aberrations in lymphocytes has been reported in workers at an open-cast uranium mine/ore processing plant in Namibia (Zaire et al 1997), but these results have been disputed (Lloyd et al 2001). An apparently increased frequency of lymphocytes with aberrations has also been reported among those living close to uranium mines, but the increase was not statistically significant and was not indicated for all classes of aberrations (Au et al 1995).

In a study of 115 smokers working in a nuclear fuel manufacturing facility who had been exposed to uranyl compounds over 1-25 years (mean lung dose ~90 mSv), a significant increase was found in frequency of chromosome aberrations in the uranyl-exposed smokers when compared with control smokers, who in turn showed a higher frequency than non-smoker controls. The increase in aberrations was attributed to the cumulative effect of smoking and exposure to uranyl compounds (Prabhavathi et al 2000).

There have been reports in the newspapers about increased frequencies of chromosomal aberrations in lymphocytes obtained from the blood of some Gulf War veterans. These reports need to be considered with caution as some chromosomal aberrations are normally present in samples of lymphocytes, and their frequency could be increased by a number of factors, including age and smoking (Tucker and Moore 1996; Sorokine-Durm et al 2000), chemotherapy, exposure to medical X-rays and radiation from other forms of medical imaging. Care is therefore needed to establish that the frequency of aberrations in Gulf War veterans is higher than that expected for individuals in the UK population and, if so, that this cannot be explained by factors other than exposure to DU.

4 Details of evidence and acknowledgements

Contributions to the report
The working group is grateful to a number of people who contributed to the preparation of the appendices to the report and their annexes. They are:

Mr Robie Kamanyire	Guy's and St Thomas' Hospital Trust
Dr Brenda Howard	Centre for Ecology and Hydrology Merlewood
Ms Stephanie Haywood	National Radiological Protection Board
Miss Katie Davis	National Radiological Protection Board
Mr Alan Phipps	National Radiological Protection Board
Mrs Tracy Smith	National Radiological Protection Board
Dr Ciara Walsh	National Radiological Protection Board
Dr Louise Ander	British Geological Survey

DU public meeting to discuss part I, on Wednesday 13 June 2001

81 people, including 12 members of Royal Society staff, attended the public meeting. The discussion panel consisted of:

Professor Brian Spratt FRS, Chairman, Royal Society working group on depleted uranium
Professor Malcolm Hooper, Chief Scientific Adviser to the Gulf War Veterans
Dr Chris Busby, The Low Level Radiation Campaign
Sir Keith O'Nions FRS, Chief Scientific Adviser, Ministry of Defence

A summary of the meeting can be found on the Royal Society web site
http//www.royal soc.ac.uk/events/DUPubMeetRevReport.pdf

Evidence obtained by the working group

The working group sought evidence from a variety of organisations and individuals, and also received a number of useful unsolicited contributions. The working group is grateful to all who participated; they are identified below.

Evidence submitted at meetings of the working group

Dr Asaf Durakovic, Uranium Medical Research Centre, Richmond Hill, Ontario, Canada
Dr Doug Rokke, Major, Medical Service Corps, United States Army Reserve

Invited attendance at meetings of the working group

Dr Chris Pickford, Harwell Scientifics
Professor Malcolm Hooper, Chief Scientific Adviser to the Gulf War Veterans
Dr Ian Ford, Condensed Matter and Materials Physics group (CMMP), University College London

Meetings with the working group

Sir Keith O'Nions FRS, Chief Scientific Adviser, Ministry of Defence, accompanied by Ron Brown, DSTL Radiological Protection Services, Institute of Naval Medicine; Professor Phil Sutton, Director Research (Corporate); Dr Campbell McCafferty; Fred Dawson, Directorate of Safety, Environment and Fire Policy; Mark Newman, Gulf Veterans Illness Unit

Policies of foreign Governments on the testing of military personnel, transmitted by science attachés based in London

Professor Salvatore Aloj	Italy
M Michel Bernier	France
Dr Wolfgang Drautz	Federal Republic of Germany
Mr James Ellis	United States of America

Evidence acquired by correspondence

Mr Ray Atherton, BNFL
Dr Keith Baverstock, WHO Regional Office for Europe
Mr Chris Busby, Low Level Radiation Campaign
Mr Ronald Brown, DSTL Radiological Protection Services, Institute of Naval Medicine
Steve Fisher, Environment Agency
Mr Dan Fahey, The Fletcher School of Law and Diplomacy, Tufts University, USA
Mr John K Jackson, Radiation Waste Management Consultant
Donald T King, Starmet
Terry A Large, Elekta
Professor Harry Lee, Ministry of Defence Gulf Veterans' Medical Assessment Programme
Mark Newman, Gulf War Veterans' Illnesses Unit, Ministry of Defence
Professor Nick Priest, Middlesex University
Brigid Rogers, Gulf War Veterans' Illnesses Unit, Ministry of Defence
Dr Doug Rokke
Professor Matthew Thirlwall, Royal Holloway University
Catherine Toque, DSTL Radiological Protection Services, Institute of Naval Medicine
Dr Eric Voice
Michael Walton, Varian

5 Glossary of terms

The technical meanings of some words as used in this report

Absorbed dose	amount of energy imparted by ionising radiation to unit mass of matter such as tissue. The SI unit for absorbed dose is joule per kilogram and its special name is gray (Gy).
Acute effects	adverse effects occurring within a short time following administration or exposure to a single or multiple doses of an agent within 24 hours. Symptoms of acute effects develop rapidly.
Aerosol fractions:	*note that these are the fractions that enter, not deposit in, these regions. Some of the respirable and thoracic fractions are exhaled without deposition; the remainder is expectorated or swallowed.*
Inhalable	the total fraction of aerosol that enters the mouth on inhalation
Thoracic	the fraction of aerosol that reaches the trachea (wind pipe) and bronchi (the two branches into which the trachea divides and that lead to the lungs). Note that the thoracic fraction will be less than the inhalable fraction.
Respirable	the fraction of the aerosol that reaches the gas exchange regions of the lung. Particles that reach this far are typically about one micron in diameter. The respirable fraction will be less than the thoracic fraction.
Alluvial	clay, silt, sand, gravel or similar material deposited by running water
Alluvial aquifer	a sandy or gravelly rock formation that holds or transmits water
Aquifer	any water-saturated stratum of earth, gravel or rock that yields supplies of groundwater in the form of wells, springs or boreholes
Biota	the animals and plants of a region
Chronic effects	adverse effects occurring at any time following administration or exposure to a single or multiple doses of an agent over a prolonged period of time (usually several months or years). Symptoms of chronic effects develop slowly over a long time period and persist or recur frequently.
Cohort	in epidemiology, a group of people whose health is followed over time
Committed dose	the dose (equivalent or effective) predicted to be received in a stated period after an intake of radioactive material, usually taken to be 50 years for workers, or up to age 70 for members of the public
Dose	general term for quantity of ionising radiation - see absorbed dose, committed dose, equivalent dose and effective dose
Effective dose	the quantity obtained by multiplying the equivalent dose to each tissue by its 'tissue weighting factor' and adding up the products. The effective dose gives a measure of the overall risk from the exposure to ionising radiation. Tissue weighting factors (listed in Part 1 Appendix I, Table 5) allow for the risk of cancer induced by radiation being greater in some tissues than in others when they receive the same equivalent dose. Effective dose is expressed in sieverts (Sv).
Epidemiology	the study of the incidence, distribution, spread and control of disease in a population

Equivalent dose	the quantity obtained by multiplying the absorbed dose by a factor (the radiation weighting factor) that allows for some types of ionising radiation being more effective in causing harm to tissue than others. The radiation weighting factor is set to one for beta particles, gamma rays and X-rays, and to 20 for alpha particles. Equivalent dose is expressed in sieverts.
Immunology	the study of the immune system, immunity and its causes and effects
Impact aerosol	a suspension of fine solid or liquid particles in gas produced on impact with a target
Ionising radiation	radiation that produces ionisation, ie the process by which a neutral atom or molecule gains or loses an electric charge. Examples are alpha particles, beta particles, gamma rays and X-rays. When these pass through the tissues of the body they have sufficient energy to damage DNA.
Kidney dysfunction	a detectable abnormality in kidney function which may or may not lead to adverse effects
Kidney failure	loss of kidney function, leading to death in the absence of appropriate medical intervention
Lymph node	small organs in the body that produce the white blood cells needed for the body to fight infection
Malignant	disease, often cancer, likely to get uncontrollably worse and lead to death
Modelling	the use of scientifically-based, quantitative, descriptions, which include known physical, chemical and biological mechanisms as far as possible, supplemented by empirical information where necessary. Models are tested as more information becomes available and they evolve as their scientific base is improved. Scientific models (not to be confused with simplistic qualitative descriptions) are valuable because (a) they bring together a large amount of established knowledge in a systematic way, (b) they can be used to check the consistency of information from different sources, and hence identify conflicts, (c) they can be used to analyse a range of scenarios in strictly comparable ways, and (d) because they allow one to estimate sensitivities to assumptions and to establish crucial gaps in data. They allow one to relate data from widely-different types of information, and they can make possible the interpretation and understanding of what is important in complex situations in which there are many inter-related factors.
Morbidity	the ratio of new cases of disease to the total population
Mortality	the ratio of deaths of individuals to the total population
Necrosis	death of a cell or group of cells whilst still part of the living body
Neonate	newborn infant
Nephrotoxic	poisonous to the kidney
Radioactivity	the property possessed by some elements, such as uranium, of spontaneously emitting energetic particles by the disintegration of their atomic nuclei
Radionuclide	an element that is radioactive
Renal	relating to, involving, or located in the region of the kidneys
Sievert (Sv)	any of the quantities expressed as equivalent or effective dose. The equivalent dose in sieverts is equal to the absorbed dose, in grays, multiplied by the radiation-weighting factor (1 Sv = 100 rem). The effective dose is the equivalent dose multiplied by the tissue-weighting factor.

Strafing attack	an attack involving machine-gun fire from low-flying aircraft at close range
Toxicology	the scientific study of the characteristics and effects of poisons
Ultrafine	particles with an average diameter of less than 0.1 micron
Yellowcake	the initial product formed from the processing of uranium ore. Uranium is extracted from the ore in solution by any one of several processes, but is then precipitated by ammonia as ammonium diuranate (ADU), and dried. The drying process often leads to partial or complete conversion to triuranium octaoxide (U_3O_8). Thus, yellowcake is a very variable mixture of ADU and U_3O_8.

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Appendix 1: The chemical toxicity of uranium

Dame Barbara Clayton, Virginia Murray and Robie Kamanyire

1.0 Background

Natural uranium (chemical symbol U) and depleted uranium (DU) are identical apart from their isotopic composition and therefore the chemical characteristics of both metal and their various compounds are the same. Hence, studies referring to the chemical toxicity of uranium are appropriate for DU.

Uranium is a naturally occurring ubiquitous heavy metal found in various chemical forms in all soils, rocks and seas. It is also present in drinking water and food. Exposure to uranium is therefore inevitable. However, the general population is most unlikely, except after the military use of DU munitions or serious accidents at fabrication plants, to be exposed to DU levels exceeding the normal uranium background levels. DU has been used for decades in medical and industrial applications, radiation shielding, counterbalance weights in aircraft and, more recently, in military armour and in kinetic energy munition rounds.

In military conflicts and their aftermath, exposure to DU will occur mostly by inhalation, ingestion, shrapnel and wound contamination. The greatest potential for exposure during military conflicts is from the inhalation of DU in aerosols produced following the impact of DU penetrators with their targets. The inhalation of such aerosols will lead to the retention of particles of DU in the lungs and their translocation to associated lymph nodes. The retention of DU within the lung and lymph nodes leads to irradiation of these tissues and the radiological consequences of the inhalation of DU have been considered in Part I of this report. Dissolution of the retained particles, or of pieces of embedded shrapnel, can lead to the exposure of tissues and organs to elevated levels of uranium with the possibility of detrimental effects resulting from both radiation and chemical toxicity. In this appendix we review what is known about the chemical toxicity of uranium and relate this information to the exposures that may occur from the use of DU munitions on the battlefield.

1.1 Toxicokinetics

Uranium has no known metabolic function. The health consequences of exposure to uranium will be dependent on the physical and chemical nature of the compound as well as the level and duration of exposure. The absorption, retention and excretion of uranium are dependent upon the chemical form of intake; the most crucial factors are the dissolution, solubility and absorption characteristics of the uranium compound.

The oxides considered to be of principal concern with the use of DU munitions are uranium dioxide (UO_2), uranium trioxide (UO_3) and triuranium octaoxide (U_3O_8). The bioavailabilities of uranium dioxide and triuranium octaoxide are relatively low (type S and M, see table 1) compared with other chemical forms of uranium. Although UO_3 and U_3O_8 are both assigned to type M, they are at opposite extremes of the range of absorption, and UO_3 is much more soluble *in vivo* than U_3O_8 (Part I, Appendix 1, Annexe A, table A5). Consideration should also be given to the ultrafine component of the aerosol (defined here as less than 0.1 μm diameter), which could represent a significant fraction of the mass and could have biological properties different from those of larger inhaled particles, possibly including much higher solubility (Ansoborlo et al 1998). The variability and uncertainties in the absorption rates necessitate caution during the calculation and interpretation of uranium biokinetic data. The variable absorption rates of different uranium oxides affect both the radiation doses and the concentrations of uranium in the organs and tissues. Thus, inhaled insoluble oxides may be retained for long periods in the lung and lymph nodes, providing the greatest levels of irradiation of these tissues, whereas inhalation of soluble oxides results in much less irradiation of the lungs and lymph nodes, but increases the potential for toxic effects as higher levels of uranium are achieved in other tissues and organs (eg the kidney).

The general population will absorb small amounts of uranium by ingestion of food, with the largest contributions coming from fresh vegetables, cereals and seafood, and water. Drinking water from mineral sources can also contain relatively high concentrations of uranium

Table 1: Classification of inhaled uranium compounds for radiological protection purposes (ICRP-68, 1994)

Type	Typical compounds
F (Fast absorption)	UF_6, UO_2F_2, $UO_2(NO_3)_2$
M (Moderate absorption)	UO_3, UF_4, UCl_4, U_3O_8
S (Slow absorption)	UO_2

See Part I, Appendix 1, Annexe A, Section A2.4 for definition of absorption Types F, M and S.

Table 2. Estimated intake, absorption, excretion and retention of uranium salts

Intake from food and water	1-5 µg per day (13-18 µg per day in uranium mining areas)	Welford and Baird 1967; Taylor and Taylor 1997; Karpas et al 1998; Roth et al 2001
Intake from air	0.0004-0.008 µg per day	Fisenne et al 1986
Absorption from gut	1-5% with a range of 0.1-6%	Wrenn et al 1985; Leggett and Harrison 1995
Absorption from lungs	About 40% of inhaled uranium salts (moderately soluble) enter the systemic circulation in a few days or weeks	Taylor and Taylor 1997
Kidneys	60-80% excreted within 24 hours of intravenous administration in animal studies In humans an average 56.2% of uranium was excreted in the urine within 24 hours	Taylor and Taylor 1997; Durakovic 1999
Bones	Uranium deposits on all bone surfaces, especially in areas of active bone growth and remodelling	Ubios et al 1991; O'Flaherty 1995
Total body content, found at post-mortem in various sample groups	56-90 µg uranium: • Skeleton: 32 µg • muscle: 11 µg • fatty tissue: 9 µg • blood: 2 µg • lungs: less than 1 µg • liver: less than 1 µg • kidneys: less than 1 µg	Roth et al 2001

and may account for a significant proportion of uranium exposure by some members of the public. Inhalation exposure is not normally a significant factor for the general population, but is relevant for occupationally exposed individuals and possibly those members of the population living near uranium mining areas or battle zones.

Inhalation of soluble compounds (eg UF_6) results in systemic absorption within days of an acute exposure. Moderately soluble compounds (eg UO_3) may remain in the pulmonary tissues and associated lymph nodes for weeks, although between 5% and 50% of the deposited material is systemically absorbed within days. Inhalation of the more insoluble compounds (eg UO_2) results in low systemic absorption and respirable particles may remain in the lungs or associated lymph nodes for years. Particles of inhaled DU oxides that are smaller than a few micrometres in diameter will deposit predominantly in the lungs. Larger particles deposit in the upper respiratory tract and will be removed by expectoration as well as by sputum and swallowing. Most of the retained material will be phagocytosed, by macrophages, and removed to the gastrointestinal tract by particle transport. Macrophages are mobile cells, rather similar to white blood cells, which may move the uranium particles to the bronchial tree, to be carried away in mucus and swallowed. Other factors, such as particle size and surface characteristics, will affect the rate of phagocytosis and the transportability of relatively insoluble material; they will also affect the absorption characteristics of the particles.

Tables 2 and 3 summarise the estimated intake, absorption, excretion and retention of uranium in humans.

The uranyl ion (UO_2^{2+}) is the most stable uranium species in solution and the most likely form to be present in body fluids. In plasma, approximately 40% of uranium is present as a transferrin complex and 60% as low molecular weight anionic complexes such as citrates and bicarbonates. The low molecular weight complexes are filtered rapidly in the kidneys and the weak transferrin complex (which is not filterable) dissociates as the low molecular weight complexes are filtered. More than 90% of soluble uranium salts injected intravenously (in animal studies) are excreted by the kidneys and less than 1% are excreted in the faeces (WHO 2001). Within 24 hours as much as 80% of injected uranium (in rats) may be filtered (Pellmar et al 1999a). The excretion is characterised by two phases, one very rapid in which 70% of the dosage is excreted in the first 24 hours, and a slow phase with a half-life exceeding several months (Berlin and Rudell 1986; Pellmar et al 1999a).

2.0 Current safety limits

Due to the paucity of human data most of the standards for occupational and environmental exposures have relied on the extrapolation to humans of conclusions derived from animal data. Occupational exposure limits were designed to maintain a concentration of uranium of less than 3 µg per gram of kidney. This limit appears

Table 3. Estimates of uptake from the gastrointestinal (GI) tract and excretion of uranium in humans (Leggett and Harrison 1995)

Study[1]	Uranium intake (µg per day)	Urinary uranium (µg per day)	Faecal uranium (µg per day)	GI uptake (central estimate %)
Masuda 1971	9.15	0.147		1.6
	5.62	0.074		1.3
	3.91	0.027		0.7
	1.77	0.006		0.3
Yamamoto et al 1974	4.51	0.14		3.1
	2.86	0.07		2.4
	1.02	0.01		1.0
	0.86	0.01		1.2
Svyatkina and Novikov 1975	23	0.6	21	2.8
	28	0.9	27	3.2
	48	1.3	46	2.7
	2310	37	2230	1.6
	2688	25	2620	0.9
Somayajulu et al 1980	30-80	2.25	57	2.2
	30-80	0.18	31	
Fisher et al 1983		0.19	24	0.8
Larsen and Orlandini 1984	1.9	0.008		0.4
Spencer et al 1990	1.9-3.7	0.01-0.08	1.9-3.6	1.5
Singh et al 1990	4.4	0.032		0.7
Limson Zamora et al 1998	3-628	1-10		3
Dang et al 1992	0.77	0.017	1.6-2.6	
Tracy and Limson Zamora 1994	30-600			1.3
Medley et al 1994	0.9-10	0.008-0.06		1.1

[1]*See Leggett and Harrison (1995) for references to individual studies*

to have been based largely on radiological considerations, rather than chemical toxicity, and was chosen to limit the radiation dose to the (then current) occupational limit of 50 mSv per year. The occupational exposure standards also took account of the health status of workers in the uranium industry (Spoor and Hursh 1973). More recent considerations of safety limits have mostly focused on the nephrotoxicity of uranium.

2.1 Modelling

The WHO uses the tolerable daily intake (TDI) approach to assess the chemical toxicity of compounds to the general public. Application of the TDI approach is often used in the assessment of chemical toxicity. The TDI approach evaluates the levels at which toxicological effects occur in various animal markers/models but does not rely on an understanding of the kinetics and spatial distribution of a specific element or compound within the body. However, biokinetic models have been extensively used in the assessment of radiological toxicity for substances such as uranium. Such models are of considerable assistance in understanding not only the radiological but also the chemical toxicity of substances such as uranium, and are used here to calculate the expected levels of uranium in the kidney from known intakes of uranium or from measurements of uranium in urine (Annexe A). The use of modelling is discussed further in Chapter 3.

The most extensively used biokinetic models for predicting the behaviour of uranium in the body are those of the International Commission on Radiological Protection (ICRP): the human respiratory tract model for radiological protection (ICRP-66 1994), the systemic

model for uranium (ICRP-69 1995) and the model for the gastrointestinal tract (ICRP-30 1979). For descriptions of these models see Part I, Appendix 1, Annexe A. Various other toxicokinetic models covering the systemic behaviour of uranium have also been used (Sontag 1986; Wrenn et al 1988; Fisher et al 1991), but these are not widely used internationally.

Whilst these models cover the distribution of uranium to all major organs and fluids, including the lungs, kidneys, liver, blood and skeleton, they do not currently specifically cover distribution to the testes or brain, tissues in which DU has been detected in rats containing implanted DU pellets (Pellmar et al 1999a). These tissues are modelled generically within the ICRP systemic model for uranium as 'soft tissues'.

2.2 Chemical toxicity

The biochemical action of all uranium isotopes is the same, because biochemical action depends only on chemical properties. Therefore the toxicities of natural, depleted and enriched uranium are considered to be identical (ATSDR 1999). The health effects from exposure to uranium have been recently reviewed (WHO 1998a,b; ATSDR 1999; Durakovic 1999; UNEP/UNCHS 1999; Fulco et al 2000; Priest 2001; WHO 2001). The health effects caused by uranium exposure, excluding effects related to ionising radiation, can be assessed using the International Program on Chemical Safety (IPCS) guidelines. These guidelines are used to derive predictive values for health-based exposure limits (WHO 1994), which are the basis of the risk estimates in the IPCS Environmental Health Criteria Document and Concise International Chemical Assessment Document (CICAD) series. In these guidelines the TDI (usually expressed as mg per kg body mass per day) is defined as 'an estimate of the intake of a substance which can occur over a lifetime without appreciable health risk'.
These intakes are based on experiments with animals, which define chronic intakes that have no observable effect (the No Observed Adverse Effect level, NOAEL) or that are the lowest intakes resulting in an observable effect (the Lowest Observed Adverse Effect Level, LOAEL). These guidelines are used to derive exposure limits for humans by reducing the NOAEL, or LOAEL, by an uncertainty factor that takes into account a number of separate factors, including the robustness of the key animal studies, and the possible differences in susceptibility of laboratory animals and humans (WHO 1994). Components of the applied total uncertainty factor are based on 'best judgement' from the available data; when no adequate data exist for a specific factor, a default value is used. Combination of these factors leads to a total default uncertainty factor of 100. Therefore, using the default uncertainty factors, a TDI for humans corresponds to the NOAEL or LOAEL concentration, derived from the key animal toxicity studies, divided by 100.

2.3 Public exposure limits

2.3.1 WHO

Ingestion: For chronic oral exposure, an initial TDI for soluble uranium, of 0.6 µg per kg per day was established by the WHO (WHO 1996) based on adverse effects in rats (LOAEL of 0.06 mg per kg per day) (Gilman et al 1998a). This has been slightly modified to 0.5 µg per kg per day based on Gilman's studies of the concentrations that result in microscopic alterations of the kidneys of rabbits (LOAEL of 0.05 mg per kg per day, divided by an uncertainty factor of 100) (Gilman et al 1998b; WHO 2001).

The ingestion of soluble uranium compounds should therefore not exceed the TDI of 0.5 µg per kg per day (35 µg of soluble uranium per day for a 70 kg adult). Insoluble uranium compounds result in lower concentrations in the kidney and the TDI is 5 µg per kg per day (350 µg of insoluble uranium per day for a 70 kg adult).

The WHO has proposed a provisional guideline for uranium in drinking water at a maximum of 2 µg per litre (IPCS 1996). This value is considered to be safe, as at this level the amount ingested by a 70 kg adult consuming two litres of drinking water per day would be about 10% of the uranium TDI (35 µg per day).

Inhalation: A NOAEL derived from several long-term inhalation studies with animals from the 1940s and 1950s approximated to 100 µg uranium per cubic metre. The application of a number of corrections and uncertainty factors suggests that inhalation of soluble or insoluble uranium compounds should not exceed 1 µg per cubic metre in the respirable fraction (WHO 2001). This corresponds approximately to a TDI for humans by inhalation of 0.5 µg per kg per day (35 µg per day for a 70 kg adult).

Studies suggest that the TDI for insoluble uranium compounds (type S) should be higher than that for more soluble compounds and a TDI of 5 µg per kg per day may be appropriate (Leach et al 1970, 1973; WHO 2001). This limit is appropriate for chemical toxicity but it would result in a total radiation dose above the radiation exposure limit for the general public (1 mSv per year), and it has been suggested (WHO 2001) that the inhalation limit for insoluble uranium compounds should be the same as that for soluble compounds (0.5 µg per kg body mass per day).

2.3.2 US Agency for Toxic Substances and Disease Registry (ATSDR 1999)

A minimal risk level for intermediate-duration ingestion has been proposed by ATSDR of 2 µg per kg per day, based on the LOAEL of 0.06 mg uranium per kg body mass per day from a study in rats (Gilman et al 1998a). A

total uncertainty factor of 30 was applied for extrapolation to humans. This minimum risk level is also considered to be protective for chronic exposures.

2.4 Occupational exposure limits

2.4.1 UK Health and Safety Executive (HSE)

The HSE has published occupational inhalation exposure standards (HSE 2000) for soluble uranium compounds:

long-term exposure limit (eight hour time average): 0.2 mg per cubic metre (200 µg per cubic metre)
short-term exposure limit (ten minute time average): 0.6 mg per cubic metre (600 µg per cubic metre)

The basis of these levels is the lack of evidence, over a period of 25 years, linking exposure to both soluble and insoluble uranium compounds, at levels well above 0.05 mg per cubic metre, with injury to the kidney. Nevertheless, the derivation of this limit is anomalous and based on radiation dose and not chemical toxicity (WHO 2001).

2.4.2 American Conference of Government Industrial Hygienists (ACGIH)

The ACGIH has established a threshold limit value of 0.2 mg per cubic metre (soluble or insoluble uranium compounds) for occupational exposures, based on a time-weighted average of eight hours. The established short-term exposure limit is 0.6 mg per cubic metre.

2.4.3 Occupational Safety and Health Administration, US Department of Labor (OSHA)

The OSHA limit for inhalation of insoluble uranium (0.25 mg per cubic metre), over a time-weighted average of eight hours, is slightly different from those of the HSE and ACGIH (0.2 mg per cubic metre). The limit for inhalation of soluble uranium salts is 0.05 mg per cubic metre.

2.4.4 WHO

Occupational exposure to soluble and insoluble uranium compounds, as an eight hour time-weighted average, should not exceed 50 µg per cubic metre (WHO 2001, Section 15.1). This limit has been suggested to overcome the contradictions between radiation and chemical exposure limits (WHO 2001, Sections 10.2, 10.4 and 12.4).

3.0 Animal Experiments

Our knowledge of the toxicity of uranium and of the exposure limits for uranium compounds has been developed largely from the substantial body of evidence from animal studies. We will not review these studies in detail, as there are large differences in toxicity between animal species and difficulties in extrapolating the results to humans. All of these animal studies, and the limited human data, establish that the primary toxic effect of uranium is on the kidney.

3.1 Inhalation

Dust particles with a diameter less than a few micrometres are generally assumed to be respirable, larger particles being trapped in the upper extrathoracic part of the respiratory tract from where they are either expectorated or swallowed.

The amounts of inhaled uranium that result in toxic effects are dependent on a number of variables, including the particle size distribution, the solubility of the uranium compound and the susceptibility of the animal species. Animal data on deposition and absorption in the lung indicate large species differences (Spoor and Hursh 1973).

Inhalation of uranium hexafluoride leads to damage of the respiratory tract but this has been attributed to the formation of hydrofluoric acid rather than an effect of uranium *per se*. Effects on the respiratory tract have been observed in some animals after inhalation of some other uranium compounds at concentrations greater than 10 mg per cubic metre, but significant effects on the lung are not generally observed after chronic inhalation of soluble or insoluble uranium compounds at concentrations less than 5 mg per cubic metre. For example, dogs, monkeys and rats can tolerate natural UO_2 (type S; table 1) aerosols of approximately 1 µm diameter and a mean concentration of 5 mg per cubic metre for periods as long as five years with little evidence of serious injury (Leach et al 1973). No evidence of chemical toxicity was found in records of body weights, mortality, haematological parameters or renal histology. Some animals were observed for protracted post-exposure periods, during which pulmonary neoplasia developed in a high percentage of dogs, two to six years post-exposure. Pulmonary and tracheobronchial lymph node fibrosis, consistent with radiation exposure and apparently dose dependent, was more marked in monkeys than dogs. There were also higher uranium concentrations in the spleen and liver of the monkeys than of the dogs at the end of the five year exposure period. The reasons for these species differences are unknown (Leach et al 1973).

Most of the animal inhalation studies are old and few, if any, have looked at the toxicity of ultrafine particles. It has been proposed that ultrafine particles are less readily taken up by macrophages and so may move to interstitial sites where they are retained. Further studies on the behaviour of ultrafine particles of uranium oxides are necessary.

The inhalation of soluble uranium compounds is considered to be more toxic to the kidneys than

Table 4: Kidney and bone concentrations of uranium observed in exposed rats and rabbits

Study	Sex/type	LOAEL (mg uranium per kg per day)	Kidney uranium (µg per gram)	Bone uranium (µg per gram)
Gilman et al 1998a	M Rat	0.060	<0.2	<1.78
Gilman et al 1998a	F Rat	0.090	<0.2	<1.78
Gilman et al 1998b	M Rabbit	0.050	0.04 ± 0.03	0.09 ± 0.05
Gilman et al 1998b	F Rabbit	0.490	0.019 ± 0.01	0.053 ± 0.004
Gilman et al 1998c	M Rabbit	<1.360	0.18 ± 0.13	0.20 ± 0.05
Gilman et al 1998c	F Rabbit	<1.360	0.18 ± 0.13	0.20 ± 0.05

inhalation of insoluble compounds (Leach et al 1970). However, dogs, monkeys and rats repeatedly exposed to relatively insoluble uranium dusts at 3-20 mg per cubic metre died of pulmonary and renal damage (Leach et al 1973). Changes in the liver of the animals were a consequence of acidosis and azotaemia resulting from renal dysfunction.

Studies with a number of animal species have established that exposure to a variety of uranium compounds by inhalation results in damage to the kidneys. The effects on the kidney range from microscopic lesions in the renal tubular epithelium at the lowest concentrations that produced an observable effect, to severe necrosis of the renal tubular epithelium at high concentrations (WHO 2001). For example, exposing mice to uranium tetrachloride dust (type M) for up to 30 days caused severe degeneration or necrosis of the renal-cortical tubular epithelium and death in animals exposed to 11 mg per cubic metre within three days. At the end of the study, moderate tubular degeneration was observed in animals exposed to 2.1 mg per cubic metre and minimal degeneration in those exposed to 0.1 mg per cubic metre (Voegtlin and Hodge 1953).

The lack of significant effects on the kidney from inhalation of uranium dusts at concentrations below 0.1 mg per cubic metre has been used to derive the safety limits for human inhalation exposure.

3.2 Ingestion

The oral dose of uranium resulting in 50% mortality of exposed animals (LD_{50}) has been evaluated for a number of species. Oral LD_{50} values of 114 and 136 mg per kg uranyl acetate dihydrate (type M) have been estimated for rats and mice, respectively, following single gavage administrations (Domingo et al 1987). Adverse effects on the kidney from a single dose of uranium have been reported to occur in rats at about 6 mg per kg (Domingo et al 1987), whereas adverse effects from chronic ingestion of uranium in drinking water occur in rats at doses as low as 1.1 mg per kg per day (Ortega et al 1989). Adverse effects involve microscopic lesions to the tubular epithelium at low doses with extensive necrosis at much higher concentrations.

Recent studies with rats and rabbits have been used to define the lowest chronic intake of soluble uranium compounds in drinking water that produces observable effects on the kidney (LOAEL, table 4). The observable effects in these animals, exposed to uranium nitrate for 91 days, were renal lesions of the tubules, glomeruli and interstitium.

These results indicate that exposure of rabbits to soluble uranium compounds for 91 days produces observable effects on the kidney at concentrations as low as 0.05 mg uranium per kg per day. In other animals, adverse effects on the kidney are observed at between one and ten mg uranium per kg per day (ATSDR 1999).

Tolerance and possibly regeneration of tubular epithelium may develop following repeated exposure to uranium, although this tolerance does not prevent chronic damage to the kidneys as the regenerated tubular epithelium cells are markedly different (Leggett 1989).

A number of studies have shown that gastrointestinal absorption of uranium is substantially greater in fasted animals, and increased absorption has also been demonstrated in neonatal rats (two orders of magnitude greater than adults) and pigs (ICRP-69 1995). In the aftermath of war the undernourishment of populations returning to war zones may increase the sensitivity of individuals to uranium toxicity, and the possibility of much greater sensitivity of neonates to uranium needs to be considered.

3.3 Dermal

There is a considerable literature on the effects of dermal exposure to uranium compounds on animals. A very thorough study was performed on rats (de Rey et al 1983). The animals received daily applications of various soluble and insoluble uranium compounds. The highly soluble compounds uranyl nitrate hexahydrate (0.5-7 g per kg body mass) and ammonium uranyl tricarbonate (7 g per kg body mass) were toxic and led to death in five days. Slightly soluble compounds like ammonium diuranate and uranium acetate were much less toxic and the most insoluble compound, uranium dioxide, was the least toxic and no changes were seen after

application to the skin. After topical application of uranyl nitrate, dense deposits were visible at the epidermal barrier level and within a few hours dense deposits were filling the intercellular spaces as well as being scattered in the cytoplasm and nuclei.

Daily applications of U_3O_8 (12 mg per day for 30 days) to the skin of rats caused epidermal atrophy. The epidermal thickness was reduced by up to 50%. Discontinuing applications for 60 days allowed only a poor recovery of 14 % of the epidermal thickness. The results of the experiment revealed that as well as epidermal atrophy there was an increase in skin permeability. The skin did not recover after a lengthy period of non-exposure (Ubios et al 1997).

3.4 Injection

Decreased glomerular filtration rate is a consistently observed outcome of acute parenteral uranium exposures in dogs, rats and rabbits. The minimum parenteral dose reported to lower the glomerular filtration rate is less than 2 mg uranium per kg body mass in rats, rabbits and dogs (Diamond 1989). Parenteral doses of 0.5-1 mg per kg in dogs, rats and guinea-pigs have caused proteinuria (Diamond 1989; WHO 2001). Glycosuria occurs at doses of 0.05 mg per kg in rats (Leggett 1989). Increased urinary alkaline phosphatase was evident in newborn rats given intraperitoneal doses of 6 mg per kg uranyl nitrate. Acute renal failure in dogs can be produced by 10 mg per kg doses of uranyl salts (Berlin and Rudell 1986).

Although it is difficult to generalise from the many studies that have been undertaken on a variety of animals, acute parenteral exposure of animals to soluble uranium compounds may produce observable effects on kidney function with exposures as low as 0.05 mg per kg, with effects on glomerular filtration rates at about 2 mg per kg and renal failure at 10 mg per kg. These values are lower than those for ingestion as the uranium is introduced directly into the blood, whereas uptake of ingested uranium to blood is only about 2%.

3.5 Implantation

A number of veterans from the Gulf War have retained DU shrapnel (see Section 4.2.5). Animal experiments have been performed to simulate the effect of embedded shrapnel using implanted DU pellets (Pellmar et al 1999a). These studies have shown that besides accumulating in the bone and kidney, uranium can accumulate within the central nervous system and testes (Pellmar et al 1999a). The implications of uranium deposition within the hippocampus are unclear, but there might be important implications for human exposures to uranium when considered in relation to the neurotoxicity of other heavy metals.

3.6 Discussion

Although there is a large literature on animal experiments, there are substantial differences between animal species resulting in considerable difficulties in extrapolating these results to humans. The availability of extensive animal data but very few human data is a well-recognised phenomenon in human toxicology and has yet to be resolved more generally. The lack of good human data on the toxicity of uranium has led regulatory authorities to define safety limits for human exposures to uranium by extrapolation from the animal data. The concentrations of uranium that lead to severe effects on the kidney are difficult to extrapolate from animal studies and are best estimated from the few reports of individuals who have been exposed to high intakes.

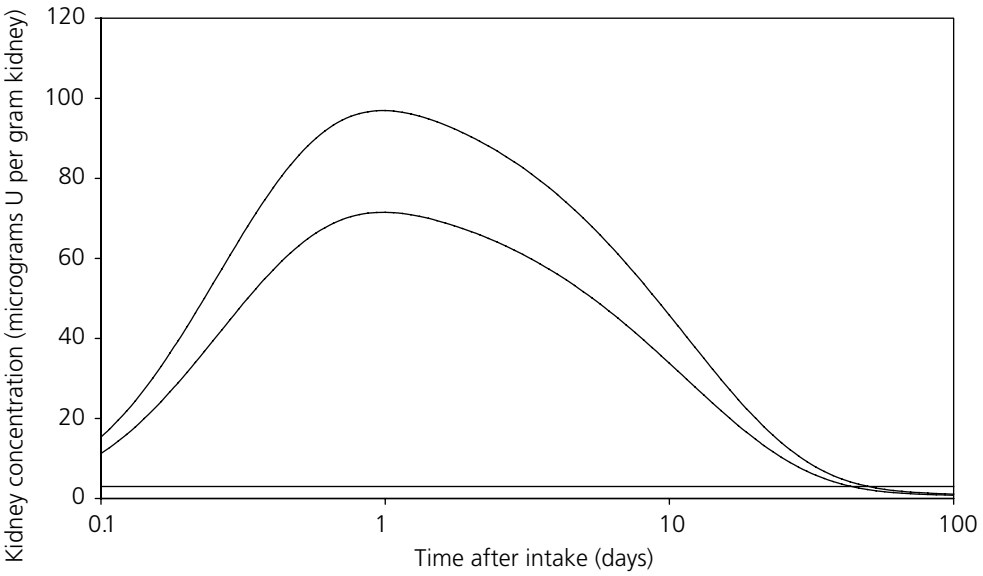
4.0 Human studies

There are relatively few reports on the toxicology of individuals exposed to high levels of uranium. Some of the most informative of these reports on human exposure, which allow the levels of uranium in urine and the predicted levels in the kidney to be related to the toxic effects, are summarised below.

4.1 Deliberate self-harm

A case of attempted suicide has been reported where a research worker deliberately ingested 15 g (146 mg per kg body mass) of uranium acetate (Pavlakis et al 1996). Initial management involved nasogastric aspiration, which recovered a moderate amount of uranium acetate, followed by gastric lavage and administration of activated charcoal. Initial investigations were unremarkable. Sixteen hours post-admission, urea was 7.8 mmol per litre and creatinine was 0.33 mmol per litre, urinalysis was normal with a pH of 5, urine microscopy showed only red blood cells (following catheter insertion), creatinine kinase was 90 IU per litre, urinary myoglobin was negative and renal ultrasound was normal, with no evidence of urinary tract obstruction. Treatment consisted of calcium disodium edetate ($CaNa_2EDTA$), 1g intravenously (IV) daily for five days, IV sodium bicarbonate to maintain urine pH above 7 and mannitol to promote a diuresis. Despite these measures the patient became progressively oliguric, and subsequently anuric, and creatinine rose to 0.89 mmol per litre at which time haemodialysis was instigated. Treatment was further complicated by the development of paralytic ileus, rhabdomyolysis (creatinine kinase peaking at 8418 IU per litre), myocarditis complicated by symptomatic atrial flutter, controlled with digoxin, anaemia (haemoglobin 8 g per dl) and liver dysfunction (ALT 192 IU per litre, AST 285 IU per litre, GGT 181 IU per litre and albumin 33 g per dl). A coagulopathy developed with an INR of 2.5 and an APTT of 50 seconds. Plasma uranium levels were 3.24 µmol per litre at two days post-ingestion; at seven days post-ingestion the whole-blood uranium level was 3.29 µmol per litre

Figure 1. Predicted uranium concentration in the kidneys following the ingestion of 15 g of uranium acetate. The two curves show the uranium concentration according to two different estimates of the fraction of the uranium absorbed from the gut to the blood (see Annexe A, Section A3.1). A solid horizontal line indicates a kidney uranium concentration of 3 µg per gram as this has been used as the basis for occupational exposure limits.



with a plasma uranium of 1.18 µmol per litre and dialysate uranium of 0.05 µmol per litre. The patient required dialysis for two weeks before renal function was sufficiently recovered. At this stage whole blood uranium was 1.07 µmol per litre and plasma uranium was 0.85 µmol per litre. The patient remained anaemic with haemoglobin levels between 7.5-8 g per dl for at least eight weeks. There was evidence of incomplete Fanconi's syndrome (a condition, which has many causes, due to a disturbance of proximal tubular function) manifesting as a renal tubular acidosis, requiring 18.5 g of supplemental sodium bicarbonate per day, glycosuria and phosphaturia. The syndrome was incomplete due to the absence of aminoaciduria. Further chelation therapy with $CaNa_2EDTA$ and calcium pentetic acid (CaDTPA) proved ineffective. Six months post-ingestion the Fanconi's syndrome still persisted, although creatinine had stabilised at 0.19 mmol per litre and haemoglobin had improved to 11.8 g per dl. There were no residual manifestations of muscle, liver or cardiac toxicity.

Using the current ICRP systemic model for uranium it was estimated from the reported measurements of uranium in urine that the maximum concentration reached was about 80-100 µg uranium per gram kidney (figure 1). The estimated level of uranium within the kidney remained above 3 µg uranium per gram kidney for about 50 days (Annexe A, Section A3.1).

This case report indicates that an acute intake of uranium that is estimated to result in a concentration of 80-100 µg per gram kidney has very serious effects on kidney function, requiring haemodialysis, and results in prolonged kidney dysfunction.

4.2 Occupational exposures

4.2.1 Acute accidental occupational inhalation of uranium compounds

Case 1: Delayed renal effects occurred after an accidental inhalation exposure to high concentrations of uranium tetrafluoride (UF_4) (absorption type M) for five minutes (Zhao and Zhao 1990). The patient was exposed to pure UF_4 powder from a clogged furnace whilst dressed in protective clothing and wearing a special gauze mask and gloves. When hospitalised 24 hours after the accident the patient was clinically well and examination of the heart, lungs, liver and kidney (including ECG and chest radiography) were normal. Six days after the accident the patient reported dizziness, nausea and anorexia. Nine days post-exposure the patient was anorexic with diarrhoea and tenesmus, with pus and blood in the stools. The symptoms resolved with a four day course of chloramphenicol. Thirty days post-exposure results of laboratory studies including full blood count, urinalysis, and renal and liver function were within normal limits. In the first 24 hours post-inhalation urinary uranium excretion was 112 µg per litre (a total of 157 µg excreted). Urinary uranium concentration increased gradually with time to a peak of about 3 mg per litre at about 60 days post-inhalation and then gradually returned to normal about three years (1065 days) post-inhalation. Renal effects were evident 78 days post-exposure, characterised by abnormal phenolsulphonphthalein (PSP) and non-protein nitrogen excretion. Amino acid nitrogen/creatinine ratio and urinary protein excretion were abnormal up to 455 days post-exposure. The patient was followed up regularly for seven years post-

exposure, and chest radiographs, electrocardiograms, liver function, thyroid function and full blood counts were normal throughout, although it is unclear whether further uranium exposure occurred (Zhao and Zhao 1990).

This intake of uranium is estimated to result in a maximum concentration of about 10 µg uranium per gram kidney, with the uranium concentration remaining above 3 µg per gram kidney for a few weeks (Annexe A, Section A3.2). The estimated peak concentration of uranium in the kidney was much lower in this case than in the case described by Pavlakis et al (1996), which is consistent with the less severe effects on kidney function.

Case 2: A 57-year-old man (employee G), who had been a uranium worker for 14 years, inadvertently removed a valve from a heated ten ton UF_6 cylinder causing the release of 3800 pounds of uranium compounds. He was immediately engulfed in a cloud of hydrolysed UF_6 (UO_2F_6) and hydrofluoric acid. The patient was observed in hospital for six days because of the risk of developing pulmonary oedema from the hydrofluoric acid exposure. Whilst hospitalised all urine was collected for uranium analysis. The patient appears to have been relatively asymptomatic and returned to work nine days post-exposure with episodic mild chest tightness. Renal function was monitored by urinalysis for protein, white and red blood cells, glucose and casts, with normal results. Urinary uranium levels peaked at 1.8 mg per litre 2.5-3.6 hours post-exposure and the total uranium excreted was 3.36 mg at 25.5 hours post-exposure (Boback 1975).

The maximum kidney concentration in this case is estimated to have been 1 µg uranium per gram of kidney at about two days post-exposure (see Annexe A, Section A3.6). The relatively low maximum kidney concentration corresponds with the absence of adverse renal toxicity. In this incident 280 employees submitted over 1000 urine samples, most of which were analysed for uranium as well as protein, sugar, white and red blood cells, and casts. There were no findings that indicated kidney damage (Boback 1975). Over 65 samples contained more than 0.1 µg uranium per litre, and six contained more than 1 µg uranium per litre (see Annexe A, Section A3.6).

Case 3: An accident at a US military facility in 1944 caused the release of an estimated 182 kg of uranium hexafluoride (UF_6). Twenty individuals were exposed in varying degrees to a mixture of steam and UF_6 or its hydrolysis products (uranyl fluoride and hydrogen fluoride). The accident resulted in two deaths and three cases of serious injury. The majority of injuries were to the eyes, respiratory tract, skin and gastrointestinal tract. In general the types of symptoms depended on concentration rather than duration of exposure, as the average exposure was calculated to be 17 seconds. The majority of initial adverse effects were probably due to thermal and hydrofluoric acid burns. However, 40-50 mg of uranium may have been deposited in the lungs of three seriously injured individuals, based on fragmentary uranium exposure data obtained shortly after exposure. The three individuals developed relatively minor renal effects (albuminuria, casts) and mental status changes believed to be in excess of what would be caused by fear. Medical and health physics assessments of two of the three seriously injured individuals, 38 years after the accident, revealed no detectable deposition of uranium nor evidence of renal damage (Kathren and Moore 1986).

Maximum kidney concentrations in these three cases were estimated to be about 1 –3 µg uranium per gram kidney (see Annexe A, Section A3.7). These levels are consistent with the relatively minor adverse renal effects.

4.2.2 Acute accidental occupational dermal exposure to uranium compounds

Case 1: A 19-year-old man received a burn to 71% of his body surface area from hot uranyl nitrate and uranium oxide (Zhao and Zhao 1990). The burnt areas were initially highly radioactive but following vigorous decontamination the five hours post-exposure radioactivity was at background levels. Urinary uranium excretion was 14 mg per litre (22 mg in total) in the first 24 hours. Two days post-exposure the patient was anorexic, with nausea and vomiting. Oliguria and proteinuria developed seven days post-exposure, and the patient was in a critical condition with severe oliguria (10 ml urine in 24 hours), pyrexia, dysphoria, coma, unspecified infection and wound effusion. The severity of burns probably caused the critical condition; burn severity is normally calculated by adding the patient's age and the body surface area burnt, and scores between 75 and 100 indicate a major injury whilst scores above 100 are potentially fatal - in this case the score was 90. Treatment was symptomatic and supportive, concentrated on maintaining adequate renal and hepatic function. The patient recovered and one month after the accident his renal and liver functions were normal. In the next 7.5 years the patient showed no physical signs of toxicity but complained of headaches, somnolence and dizziness. Occasionally his leucocyte and platelet counts were slightly low (Zhao and Zhao 1990).

It was estimated that the kidney concentration might have reached a maximum of about 35 µg uranium per gram kidney, depending on the assumed rate of dermal absorption. If the rate of absorption is reduced, the estimated maximum concentration is lower, but the concentration remains elevated for longer. It was also estimated that the concentration remained above 3 µg uranium per gram of kidney for about 40 days (Annexe

A, Section A3.3). This report suggests that a peak kidney uranium concentration of about 35 µg per gram can cause serious kidney dysfunction, but the extensive burns sustained by this individual would almost certainly have contributed to his critical condition.

Case 2: Butterworth (1955) reported another case of dermal exposure to hot uranium compounds. In this case the predicted maximum kidney concentration was about 3 µg uranium per gram ten days after the accident, with the level remaining above 1 µg per gram for 20-30 days (Annexe A, Section A3.5). Some adverse effects on the kidney (albuminuria) persisted until the beginning of the third week after exposure.

4.2.3 Chronic occupational exposure during uranium milling

One study of the renal function of uranium mill workers chronically exposed to 'yellowcake'[1] revealed renal tubular dysfunction (mild proteinuria, aminoaciduria) when compared with a control group of cement workers. Data from this study are indicative of reduced renal absorption capacity in the proximal renal tubules (Thun et al 1985). The mill workers had significantly higher excretion of β_2-microglobulin (BMG) and various amino acids, although the upper limit of normal BMG excretion was not exceeded. Interestingly, the clearance of BMG generally increased with time depending upon how many years the workers had been in the yellowcake drying and packaging area.

In these workers 21% of their urine samples contained more than 30 µg uranium per litre and some individuals excreted about four times this level. Assuming an output of 1.5 litres of urine per day, the workers exceeding this level of urinary uranium would have had at least 0.25 µg uranium per gram kidney (Annexe A, Section A2.2) and the highest level would have been about 1 µg per gram. The signs of kidney damage in the workers are therefore consistent with the view that chronic exposure that leads to concentrations less than 3 µg uranium per gram kidney are nephrotoxic. The lack of data on the uranium levels in urine for individual workers in relation to their kidney function tests precludes a more precise assessment of the uranium levels causing toxicity.

A significant feature of this study is that the tests showing enhanced levels of indicators of kidney dysfunction appear to have been carried out more than a year after the elevated levels of uranium exposure.

4.2.4 Chronic occupational inhalation

A study performed in the 1940s where 31 uranium workers were examined after year-long inhalation exposure to dusts of uranium (VI) oxide, uranium peroxide and uranium chlorides (at concentrations that at times reached 155 mg uranium per cubic metre) did not reveal any symptoms or signs of chronic poisoning (Clark et al 1997). It is likely that the methods of occupational health monitoring used during this study would be considered inadequate in comparison with current standards.

4.2.5 Retained DU fragments

In a cohort of 33 US soldiers wounded in the Gulf War, 15 having retained shrapnel, examination revealed no evidence of a relationship between urinary uranium excretion and renal function three years after the injuries. The clinical assessment of renal function was satisfactory with the measurement of urinary protein, creatinine, glucose and BMG. However, the study did not investigate the specific nature of the retained shrapnel, which might have been contaminated with alternative heavy metals, nor was a thorough heavy metal urinalysis carried out (Hooper et al 1999).

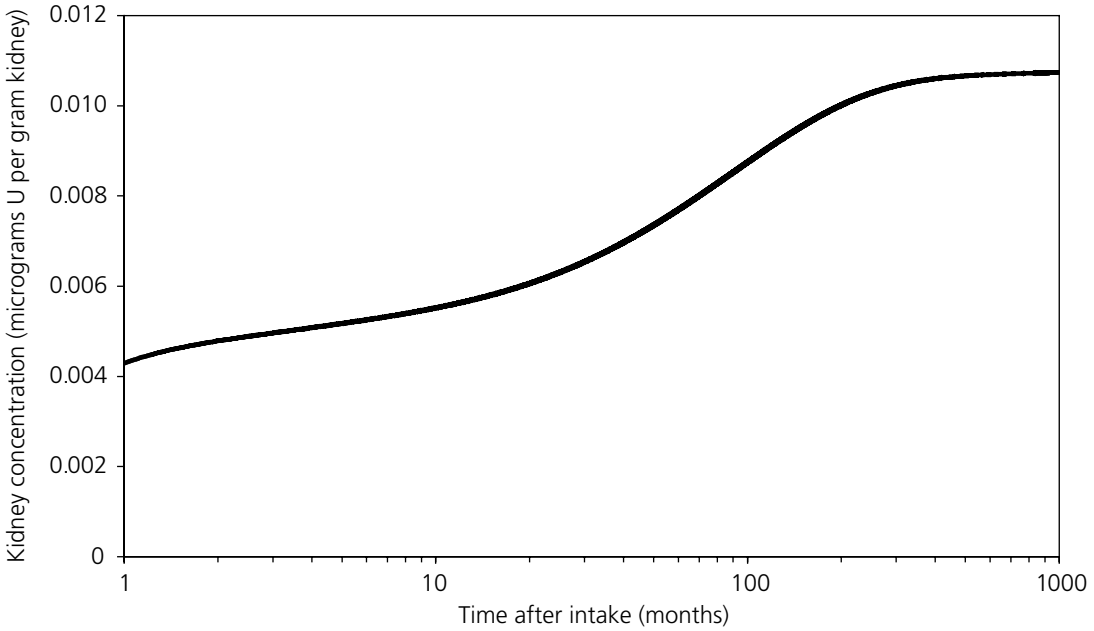
From the data of Hooper et al (1999) and McDiarmid et al (2000), the highest urinary excretion among the veterans with retained DU shrapnel was about 60 µg uranium per day (converted from µg per gram creatinine in the above publications, assuming the excretion of 2 g of creatinine per day). Most of the uranium entering the blood is excreted in the urine and the rate of uptake of uranium to the blood is approximately equal to the urinary excretion rate. From figure 2 an uptake rate of 1 µg uranium per day gives a kidney uranium concentration of 0.0056 µg per gram kidney at one year and 0.0090 µg per gram kidney at ten years. For the soldier with the highest level of uranium entering the blood (60 µg per day) from DU shrapnel, we therefore predict about 0.3 µg uranium per gram kidney at one year and about 0.5 µg uranium per gram kidney at ten years. Measurements between 1993 and 1995 (Hooper et al 1999) showed an average urinary excretion rate of about 10 µg per day for the soldiers with retained uranium, which would be predicted to result in 0.06 µg uranium per gram kidney at one year and 0.1 µg uranium per gram kidney at ten years (Annexe A, Section A2.3).

4.3 Volunteer studies

Inevitably, volunteer studies mostly involve intakes of relatively low amounts of uranium, and typically have been used to understand the biokinetics of uranium in humans, rather than the levels that are toxic to humans. Butterworth reported a volunteer who ingested a

[1] 'Yellowcake' is the term given to the initial product formed from the processing of uranium ore. Uranium is extracted from the ore in solution by any one of several processes, but is then precipitated by ammonia as ammonium diuranate (ADU) and dried. The drying process often leads to partial or complete conversion to triuranium octaoxide (U_3O_8). Thus, yellowcake is a very variable mixture of ADU and U_3O_8 (Edison 1994).

Figure 2. Predicted concentration of uranium in the kidney from the constant uptake into the blood of 1µg uranium per day.



relatively large amount (1 g of uranyl nitrate in 200 ml water) of a soluble uranium compound. The volunteer developed acute nausea, vomiting and diarrhoea within a few hours of administration but within 24 hours recovery was complete. Within three hours of ingestion the urinary uranium concentration was 8000 µg per litre, but within 60 hours the levels had fallen below 100 µg per litre. Albuminuria only occurred on two occasions when the urine uranium concentration was at its highest. The approximate amount of uranium excreted during the first seven days was 2.5 mg. The excretion on the first day was approximately 15 times greater than that on the second day. Presuming renal excretion of 66% of the absorbed amount, it would suggest that at least 1% of the oral dose was absorbed even ignoring the unknown amount of uranium in the vomit and faeces (Butterworth 1955).

The maximum kidney uranium concentration in this case is estimated to be about 1 µg per gram (Annexe A, Section A3.4). The slight effect on kidney function at the peak of uranium excretion indicates that slight adverse effects on the human kidney can be observed at this kidney uranium concentration.

In the 1950s studies of the radiological treatment of brain tumours involved determining a tolerable intravenous dose of uranium (Luessenhop et al 1958). Five patients with malignant brain tumours were selected for the study (table 5). The patients were comatose or semi-comatose, but otherwise were healthy with no other disease. Uranium nitrate was injected intravenously at doses between 0.1 and 0.28 mg uranium per kg. There were no consistent or marked changes in vital signs (blood pressure, pulse, temperature or respiration) following the injections. Liver function tests and haematological studies also remained unchanged following the injections. Some patients developed short periods of oliguria and urinalysis showed the presence of hyaline casts and elevated levels of protein and catalase, indicating some kidney dysfunction.

The estimated kidney uranium concentrations in these cases range from 1 to 6 µg per gram (Annexe A, Section A3.9), which is consistent with the presence of significant kidney dysfunction in some of these patients.

4.4 Environmental exposures

A study (Limson Zamora et al 1998) compared two Canadian communities where one was supplied with mains water (less than 1 µg uranium per litre) and another's drinking water contained between 2 and 780 µg uranium per litre. The range of total daily uranium intake through both food and drinking water was 3–570 µg, with the percentage through intake of water varying between 31% and 98%. Renal toxicity markers (glucose, creatinine, total protein and BMG) as well as cell toxicity markers (eg alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), N-acetyl-β-D-glucosaminidase (NAG) and lactate dehydrogenase (LDH)) were monitored. Increased urinary glucose, BMG and ALP were evident in the high-exposure group. Glucose excretion increased with increasing daily uranium intake. Urinary glucose was found to be significantly different and positively correlated with uranium intake for pooled data. Increases in ALP and BMG were also positively correlated with uranium intake for pooled data. The results suggest that at total daily intakes between 0.2 and 9 µg per kg the chronic

Table 5. Intakes and kidney effects of uranium nitrate injected intravenously

Patient	Age (years)	Sex	Weight (kg)	Uranium dose (mg)	Renal function and urinalysis results	Kidney uranium (µg per gram)[1]
1	26	M	55.9	5.5	no abnormalities	1.8
2	47	M	57.4	5.9	protein, catalase	2
3	34	M	60.0	4.3	no abnormalities	1.4
4	63	F	67.7	11.2	casts, protein, catalase, urea, non-protein nitrogen, creatinine	4
5	39	M	55.9	15.8	casts, protein, catalase, non-protein nitrogen, urea, creatinine	6

[1]*The estimated maximum kidney uranium concentrations in these cases range from 1 to 6 µg per gram (Annexe A, Section A3.9).*

ingestion of uranium in drinking water affects renal function (manifested as increased urinary glucose, ALP and BMG), and that the proximal tubule rather than the glomerulus is the predicted site of injury. The observed effects suggest subclinical toxicity that will not necessarily lead to kidney failure or overt illness in individuals who are exposed to high levels of uranium in drinking water. It may, however, be the first step to the development of renal failure.

From figure 2, after one year of constant uptake to blood of 1 µg per day, the level of uranium is predicted to reach 0.0056 µg per gram kidney, and after 50 years it would reach 0.011 µg per gram kidney. For uranium in soluble form it is generally assumed that for adults 2% of ingested uranium is absorbed into the blood (ICRP-69 1995). Thus it is predicted that these levels would be reached from constant ingestion of 50 µg per day of soluble uranium. These values can be scaled up to estimate the levels of uranium in the kidneys of the individual with the highest average daily intakes of soluble uranium (570 µg of uranium per day) in the study of Limson Zamora et al (1998). After one year of chronic exposure, the level of uranium is predicted to reach 0.06 µg per gram kidney and after 50 years of daily exposure it would reach 0.13 µg per gram kidney (Annexe A, Section A2.1). Concentrations substantially below 3 µg per gram may therefore lead to significant toxicity to the human kidney. It is pertinent to note that there may be significant differences in the concentrations of other toxic metals between municipal water and water from private wells. However, the positive correlation between kidney dysfunction and the level of uranium intake gives confidence that the observed effects were due to the intakes of uranium.

The slight effects on kidney function seen in individuals with chronic uranium intakes that are estimated to result in kidney concentrations of about 0.1 µg per gram kidney are slightly inconsistent with the lack of any reported signs of kidney dysfunction in soldiers with retained DU shrapnel, where ten years after the Gulf War levels of kidney uranium in some soldiers are expected to be more than five times this level.

4.5 Post-mortem studies

Clinical post-mortem studies of occupationally exposed workers indicate significant amounts of uranium in the lung tissues, suggesting that inhalation is an important source of accumulation (Kathren et al 1989; WHO 2001). In autopsies of chronically exposed individuals, uranium has been observed in the skeleton, liver and kidneys in the average ratio of 63:2:8 (Kathren et al 1989). Variations in this ratio are common and are dependent on the pattern and nature of exposure (ATSDR 1999).

Analysis of post-mortem wet lung tissue, from the general population, in New York, Chicago and San Francisco revealed levels of 0.001 µg per gram with a range of 0.0007-0.003 µg per gram (Welford and Baird 1967). A comparison of kidney tissue obtained at autopsy from seven uranium workers and six control subjects with no known exposure to uranium showed that the groups were indistinguishable (Russell et al 1996). The uranium concentrations in the kidneys of the seven uranium workers ranged between 0.0004 and 0.25 µg per gram.

A histological examination was made of kidney tissue sections obtained at autopsy from seven persons exposed to uranium and six controls (persons not exposed). This was a blind study, and the pathologist was unable to identify the subjects who had been exposed to uranium. It was concluded that the chronic low-level exposure, which was an order of magnitude lower than the accepted permissible occupational level of 3 µg uranium per gram, did not induce identifiable permanent tissue damage (Russell et al 1996).

Studies of aborted human foetuses have shown a uranium concentration of about 10% of that in their mothers (Weiner et al 1985). These studies, and those with animals (Sikov and Mahlum 1968 McClain et al 2001), indicate that exposure to high levels of DU prior to, or during, pregnancy will lead to increased levels of uranium in the foetus.

Table 6a: Acute human exposures to uranium resulting in effects on the kidney.

Route of exposure	Chemical form	Subject(s)	Intake (mg)[2]	Kidney uranium (µg uranium per gram kidney)[1]	Renal effects[3]	Outcome	Reference
Ingestion	Acetate	Adult male	8500	100	+++	Residual renal dysfunction (incomplete Fanconi's syndrome) six months post-exposure	Pavlakis et al 1996
Dermal (burn)	Nitrate	Adult male	130	35	+++	Renal and liver function normal one month post-exposure	Zhao and Zhao 1990
Inhalation	Tetrafluoride (UF_4)	Adult male	900	10	++	Biochemical indication of renal dysfunction up to 18 months post-exposure	Zhao and Zhao 1990
Injection	Nitrate	Adult male Adult female (terminally ill)	10	5	++	Pyelonephritis and changes to epithelium of convoluted tubules at post-mortem	Luessenhop et al 1958
Dermal (burn)	Nitrate	Adult male	10	3	++	Albuminuria persisted for three weeks post-exposure	Butterworth 1955
Inhalation	Ore concentrate[4]	Adult male[5]	200	3	–	No evidence of renal dysfunction for at least one year post-exposure	Boback 1975
Injection	Nitrate	Three adult males (terminally ill)	5	2	+	Casts in collecting tubules at post-mortem	Luessenhop et al 1958
Inhalation	Hexafluoride (UF_6)	Three adult males	50–100	1–3	+	Complete recoveries within 10-21 days No evidence of adverse effects 38 years post-exposure	Kathren and Moore 1986
Ingestion	Nitrate	Adult male	470	1	+	Complete recovery within 24 hours	Butterworth 1955
Inhalation	Hexafluoride (UF_6)	Adult male[5]	20	1	–	Patient returned to work nine days post-exposure	Boback 1975

BMG, $ß_2$-microglobulin; ALP, alkaline phosphatase.
[1]Estimated maximum kidney concentration (µg uranium per gram kidney); for details see Annexe A.
[2]Estimated uranium intake.
[3]Renal effects:
+++ severe clinical symptoms (eg oliguria, anuria, rhabdomyolysis, acute renal failure)
++ protracted elevation of indicators of renal dysfunction (eg albuminuria, glycosuria, casts)
+ transient elevation of indicators of renal dysfunction (eg non-protein nitrogen, phenolsulphonphthalein, BMG, ALP)
- biochemical tests on urine negative.
It should be noted that the investigations of renal function have greatly improved over the last 40 years, therefore subtle effects on renal function may not have been noted in the older references.
[4]Mixture of diuranates (eg sodium and ammonium) and uranium oxides.
[5]Boback gives information on three more subjects exposed to ore concentrates, and on six more exposed to UF_6, for whom urinary excretion of uranium was similar to, but lower than, these cases. Biochemical indicators of kidney dysfunction were negative in all.

4.6 Summary of human studies

Table 6 shows summary data on human exposures to elevated levels of uranium. The limited human studies indicate that effects on the kidney can be observed following chronic intakes that lead to kidney uranium concentrations as low as 0.1 µg per gram, or acute intakes that transiently result in peak concentrations of about 1 µg per gram. Very severe effects, which would probably be lethal in the absence of appropriate medical intervention, appear to occur after acute intakes that lead to concentrations above about 50 µg per gram kidney.

Table 6b: Chronic human exposures to uranium resulting in effects on the kidney.

Route of exposure	Chemical form	Subjects	Markers of renal dysfunction	Kidney uranium (µg uranium per gram kidney)[1]	Reference
Inhalation	Yellowcake	27	Elevated BMG	up to ~1	Thun et al 1985
Subcutaneous or intramuscular	Uranium metal	15	No abnormalities	up to ~0.5	Hooper et al 1999
Ingestion	Drinking water	30	Glycosuria, elevated BMG and ALP	up to ~0.1	Limson Zamora et al 1998

[1]*Estimated maximum kidney concentration (µg uranium per gram kidney); for details see Annexe A.*

5.0 Target organs

5.1 Renal effects

The toxic action of uranium on the kidneys is not fully understood. In a normal kidney, urine is filtered through the glomeruli and passes into the tubules where 60% of the glomerular filtrate is reabsorbed, mainly by active sodium transport. The surfaces within the proximal tubules contain specific carrier systems for the absorption of sugars, amino acids and phosphate. Calcium, uric acid and various trace metals are also absorbed. The tubules are rich in mitochondria, and oxidative metabolism involving glutamine, lactate and fatty acids occurs in the proximal tubules. Blood flows through the kidneys at the rate of 1000-1200 ml per minute in an adult human male. After the age of 35 years, the glomerular filtration rate falls by about 10% every ten years. A total of 98% of the total filtered load of α-amino acids is reabsorbed in the proximal tubules. In healthy individuals, the protein excretion in urine is only 20-35 mg of albumin each day.

If the tubules are damaged, there is increased excretion of small protein molecules, including BMG and retinal-binding protein, that are normally used as measures of this type of damage. There may also be excretion of amino acids and other compounds. This type of damage is also seen, more severely, with cadmium, lead, mercury and some organic solvents.

In renal diseases of occupational origin, modern techniques can detect microgram amounts of low molecular weight proteins following subtle renal tubular injury (Wedeen 1992). Toxin-induced acute tubular necrosis rarely results in permanent kidney damage except in cases observed with certain salts of mercury, chromium and uranium that produce acute tubular necrosis by direct damage to renal tubule epithelial cells.

It should also be noted that renal failure or damage usually produces no clinical symptoms until two-thirds of kidney function has been lost. In the last 20 or 30 years more sensitive tests of renal tubular function have been introduced into routine laboratory services and minor tubular injury may be detected by the measurement of low molecular weight proteins in urine. Their presence, however, does not necessarily indicate that renal failure will develop in later life (Bernard and Lauwerys 1991).

In experimental animals, uranium-induced renal injury becomes evident soon after exposure, as changes in the proximal tubules. It is thought that the binding of uranium to the brush border membrane in the distal portion of the proximal tubules may cause reduced reabsorption of sodium and consequently reduced reabsorption of glucose, proteins, amino acids and water. If the pH of the tubular urine is low, some uranium will be reabsorbed in the tubules, whereas at high pH small amounts of uranium will be retained in the tubular walls. There is also the possibility of complex formation between uranium ions and proteins on the tubular walls, which would impair or damage the plasma membranes. Later structural damage to the plasma membrane may cause extensive changes in membrane transport and permeability. Mitochondrial dysfunction may occur due to changes in the intracellular environment after alterations in plasma membrane permeability. Declines in cellular energy production due to altered mitochondrial function may lead to alterations in active transport mechanisms across the renal tubular membranes and to diminished capacity to repair the affected plasma membranes (Leggett 1989).

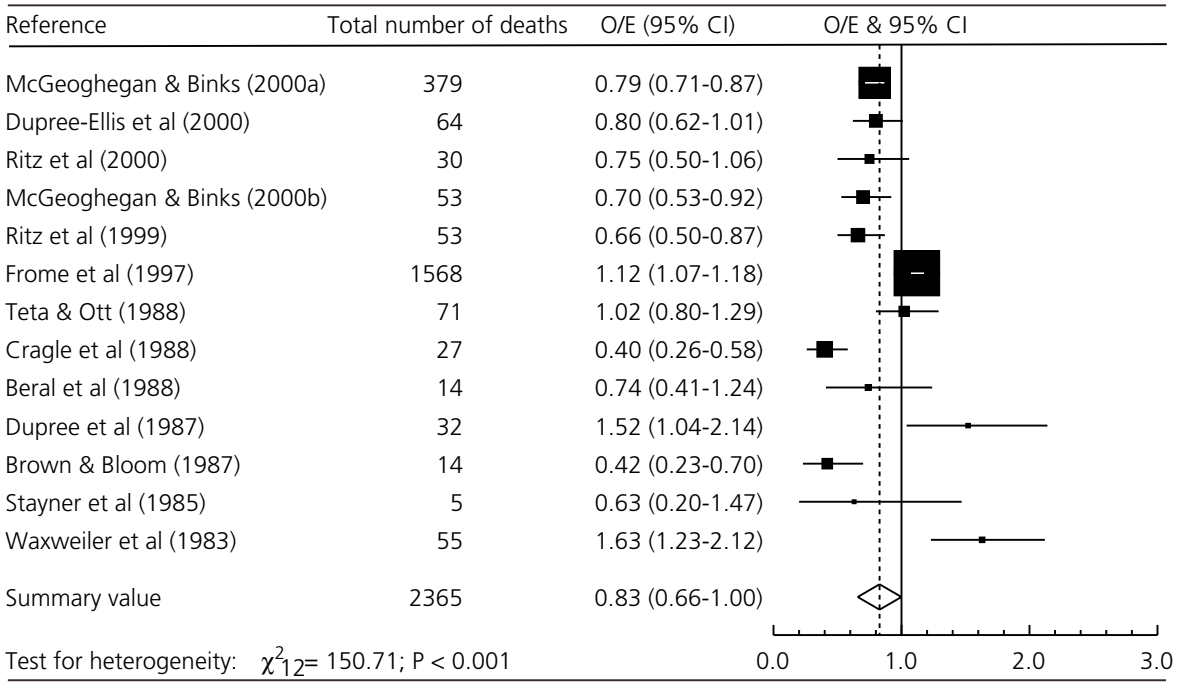
Increases in renal tubular carcinoma have been observed in mice after injection of 40-197 kBq ^{233}U per kg body mass (Ellender et al 2001). However, uptakes to blood of even 1 kBq per kg of the much less radioactive DU would be lethal to humans due to chemical toxicity.

5.2 Non-malignant respiratory disease

Workers in the uranium industry and underground uranium miners have been chronically exposed to uranium dusts but there are few data on rates of non-fatal respiratory disease. Mortality from non-malignant respiratory diseases in uranium workers is summarized in figure 3.

Overall the number of deaths observed in the combined studies was 17% fewer than the number expected from general population rates, although in three individual studies (Waxweiler et al 1983; Dupree et al 1987; Frome et al 1997) the numbers of deaths observed were significantly

Figure 3. Ratio of observed number of deaths from non-malignant respiratory disease in uranium workers compared to that expected in the general population.



greater than the numbers expected from general population rates, by factors of 1.12, 1.52 and 1.63, respectively. Some studies therefore suggest a significant increase in mortality from non-malignant respiratory disease among uranium workers (NECIWG 2000), but in interpreting these results it must be remembered that mortality from many respiratory diseases (eg chronic bronchitis) is determined largely by smoking habits, and other toxic exposures may be present. However, the findings do rule out the possibility of large increases in respiratory deaths among uranium workers.

Occupational exposure to a number of metal dusts or fumes has been associated with several non-malignant lung diseases, including pneumonitis, pulmonary oedema, acute tracheobronchitis, obstructive lung disease, metal fume fever and occupational asthma (Nemery 1990; Kelleher et al 2000). However, uranium is not one of the metals that have been associated with these types of lung disease.

Interstitial pulmonary fibrosis (scarring and thickening of lung tissue) leading to shortness of breath and eventual cardiopulmonary failure has been observed in uranium miners but has been attributed to alpha-particles from highly radioactive radon progeny and possibly silicates (Archer et al 1998).

Pulmonary damage has been observed in animals after long-term inhalation of some uranium compounds at concentrations above about 5 mg per cubic metre (Leach et al 1973; Spoor and Hursh 1973). Increases in respiratory frequency were observed after latent periods of one year or more in dogs inhaling aerosols of $^{239}PuO_2$ at levels above 0.33 kBq per kg initial lung burden, resulting in accumulated lung doses over seven years of 2.3 Gy and above (Muggenburg et al 1988). The clinical signs were confirmed by cardiorespiratory function tests six years later. In other studies in dogs by the same group (Muggenburg et al 1999), the lowest accumulated dose to produce pneumonitis was found to be 6.3 Gy following initial lung burdens of 1.0 kBq $^{239}PuO_2$ per kg or higher. The pneumonitis progressed into lung fibrosis which later was lethal, and the mean time to death was 3.9 years after the range of initial lung burdens used of 0.19-30 kBq per kg. The levels of accumulated absorbed dose are equivalent to several tens of sieverts using a radiation weighting factor of 20, and hence are above, but of the same order of magnitude as, the estimated worst-case Level I lung equivalent dose of 9.5 Sv accumulated over 50 years in the modelled human situation with inhalation of 5 g of DU.

Some soldiers on the battlefield might receive inhalation intakes of DU oxides that are very substantially greater than the daily intakes that occurred in uranium workers, and the increased risks of lung cancer in such soldiers have been considered (see Part I and Chapter 3 of Part II). The nature of the inhalation intakes (particle size, presence of a significant microfine component, solubility, etc) is also likely to be different in the industrial setting (and in animal experiments) compared with the battlefield, which increases the difficulty in assessing the respiratory toxicity of inhaled DU. Acute respiratory effects would not be unexpected following the inhalation of large amounts of dense DU aerosols (for example, for any survivors in a tank struck by a DU penetrator or those working for protracted periods in contaminated vehicles).

It is unclear whether large inhalation intakes of DU would lead to sufficient alpha-particle irradiation of the lungs to cause significant fibrosis, but the possibility perhaps exists for worst-case Level I or II intakes, as the radiation doses are not very much lower than those at which pulmonary effects occur in dogs, and there is evidence that dogs may be about two-fold less sensitive to radiation-induced pulmonary damage than humans (Poulson et al 2000).

Long-term respiratory effects for soldiers who inhaled smaller amounts of DU from aerosols (most Level II and all Level III inhalation exposures) are considered unlikely.

5.3 Endocrine effects

In a detailed study of Gulf War veterans no effect of uranium was found on the semen of those with either high or low uranium exposures (WHO criteria) seven years after the war (McDiarmid et al 2000). Prolactin, follicle-stimulating hormone, luteinizing hormone and testosterone levels in urine were also measured. The results showed no differences between the high- and low-exposure groups. However, when the results were ranked and stratified with low and high uranium exposure groups, there was a seven-fold difference in urinary uranium concentrations between low and high prolactin levels: 1.66 vs. 12.47 µg per gram creatinine. Although this was considered to be an endocrine effect, the result for prolactin might be due to proximal tubular damage. Low molecular weight hormonal excretions are well recognised in renal physiology and are not considered an indicator of endocrine abnormalities (Ramirez et al 1978; Maack et al 1979).

5.4 Haematological effects

There is very little information on the haematological effects of uranium intakes in humans. In an extensive study of Gulf War veterans, seven years after potential DU exposure, haematological parameters were studied in high and low 'spot' uranium groups. Tests included white blood cell counts, measurement of haematocrit and haemoglobin levels, and counts of platelets, lymphocytes, neutrophils, basophils, eosinophils and monocytes. There was no statistical relationship between the results in high- and low-exposure groups but there was a non-significant trend toward higher eosinophil counts in the high uranium exposure group (McDiarmid et al 2000). Transient anaemia was observed in the individual studied by Pavlakis et al (1996) who attempted suicide by ingesting 15 g of uranium. However, animal studies indicate that significant haematological effects are generally only observed after chronic exposures to relatively large daily intakes of uranium (Ortega et al 1989).

5.5 Neurocognitive effects

DU has been found in the hippocampus of rats with DU implants and this has raised the possibility of adverse neurocognitive effects in veterans exposed to high levels of DU (Pellmar et al 1999b). The literature on uranium and neurotoxic effects is sparse and not generally described in depth. A statistical relationship was evident between uranium levels and poorer performance on computerised tests that assessed performance efficiency, when Gulf War veterans, who had or had not been exposed to DU, were compared (McDiarmid et al 2000). There was no substantial relationship between urine uranium and cognitive test performance in 30 Gulf War participants (mean age 28 years). However post-traumatic stress disorder may be an important clinical factor, for which no suitable control groups were available for comparison (Kane et al 1997). Overall it is currently impossible to come to any firm conclusions about the possibility of substantial intakes of DU leading to neurological disease.

5.6 Bone effects

The literature on humans is sparse and most of the research on uranium-induced changes in bone has been conducted on animals. Significant amounts of uranium can be found in bone a considerable time after exposure. Bone is thus considered a critical organ in chronic exposure to uranium. In the rat, in both acute and chronic intoxication, it causes a decrease in bone formation and may increase bone resorption (Ubios et al 1991). Since there are differences in the handling of uranium by different species, animal experiments will not be discussed further. There are no human data that can be used to predict whether large exposures to DU on the battlefield could have effects on bone.

Finkel (1953) found an elevated and dose-dependent incidence of osteosarcoma in mice after injection of ^{233}U (a 31% incidence after injection of 925 kBq per kg body mass). These very high levels of activity are of little relevance to DU, as huge intakes of the much less radioactive DU would be required to achieve this activity, which would certainly be lethal due to chemical toxicity.

It has been suggested that uranium complexed at physiological pH (~7.4) should behave similarly to the alkaline earths, making the skeleton the principal site for uranium accumulation. The greatest numbers of uranium measurements in tissues, from environmentally exposed individuals, have been in bone, followed by kidney, blood, lung, muscle, fat and other tissues. A literature review (Fisenne et al 1988) has revealed data on uranium concentrations in human bone from 12 countries. The data are normalised to dry ash, and using the geometric mean of 7.3 µg of uranium per kg of ash yields an estimate of the median skeletal burden of 20 µg uranium; using the arithmetic mean of 11 µg of uranium per kg of ash yields an average skeletal burden of 30 µg uranium. The review also revealed sparse data on the concentrations of uranium within soft tissues such as lung, liver, kidney and muscle. The authors propose that further studies are necessary to reveal

whether muscle and fat or bone marrow form major reservoirs for uranium in the human body and similarly whether uranium accumulates in the brain. An answer to the latter would be important in view of possible neurocognitive effects (see above). Similarly, uranium crosses the placenta and the effects of maternal exposure to DU on skeletal development in the foetus may also need to be considered.

5.7 Immunological effects

To the best of our knowledge there are no published studies of the effects of DU on immune function. However, it is unlikely that exposure to DU on the battlefield will lead to major changes in serum immunoglobulins, complement, or in B or T lymphocyte numbers or function (Personal communication, Professor Freda Stevenson). Kalinich et al (1998) have studied the effect of DU-uranyl chloride at concentrations up to 100 micromolar on the viability of rodent thymocytes, splenocytes and macrophages, and on human T-cell leukaemia and B-cell lymphoma cell lines, and a mouse macrophage cell line. Effects were only observed with macrophages that showed a dose-dependent loss of viability, appearing to undergo apoptosis, and had a reduced ability to phagocytose bacteria.

Following inhalation of DU aerosols, the deposition of particles within respiratory lymph nodes may cause the death of traversing lymphocytes due to irradiation by alpha-particles, but this is unlikely to lead to any substantial reduction in the ability of the body to combat infection (see Chapter 3 where possible radiological effects on the immune system are discussed further).

Whether there could be slight effects on immune status in soldiers with high intakes of DU is less easy to evaluate. Korényi-Both et al (1992) have described a pneumonitis (Al Eskan disease) that they associate with exposure to the very fine sand particles (0.1-0.25 µm diameter) present in the Persian Gulf. They have proposed that ultrafine sand particles can be pathogenic, not simply due to acute silicosis but to allergic hypersensitivity to the ultrafine sand associated with pathology of the immune system. The proposed immunosuppression has been suggested to be a contributory cause of Gulf War Syndrome (Korényi-Both et al 1997). Whether exposure to ultrafine sand can lead to immunosuppression is unclear but the possibility adds to the list of potentially toxic exposures, which include multiple vaccinations, squalene in vaccine components, antidotes to nerve agents, pollution from oil well fires, pesticides and rodenticides, organic solvents and perhaps DU, that together may contribute to the symptoms seen in veterans of the Persian Gulf War.

Effects on the immune system might be revealed by an increased incidence of infections, but subtle effects may not be detected. Disorders of immunity could also lead to autoimmune disease, or an increased incidence of cancer due to reduced immune surveillance, both of which are only likely to become evident in later life, and cannot be easily predicted at an early stage.

The immune system includes a wide variety of interacting elements, which generate antibody and cellular responses. In an individual, the immune status will vary according to exogenous influences, especially infection. It is difficult, therefore, to know which measurements to apply to determine if there is an acquired defect in those heavily exposed to DU aerosols. One useful marker of immune activity is C-reactive protein (CRP) (Du Clos 2000). Serum CRP is a classical acute phase protein, which may be raised 1000-fold in response to infection, ischaemia, trauma, burns and inflammatory conditions. Production is initiated by a cytokine (IL-6) and it occurs rapidly following infection. CRP is an indicator of activation of the innate immune response, and is increased in several clinical conditions, including cardiovascular disease (Danesh et al 2000). However, in normal adults a raised level is likely to be associated with persistent bacterial infection. Failure to clear infection is an indicator of immunodeficiency.

Immunodeficiency can also be associated with a failure of cytotoxic T cells to control endogenous viruses. It is possible to monitor a decline in the ability of the immune system to regulate persistent viruses, such as Epstein-Barr virus (EBV), by measuring viral load in the blood using a quantitative polymerase chain reaction (PCR) (Ohga et al 2001).

In summary, in normal adults, measurement of CRP presents a simple and economical way of assessing a failure to control bacterial infection. Although not a specific test, normal levels would argue against damage to the immune system, and could be used as a measure of immunotoxicity. Measurement of EBV load is a more expensive test, and less widely used. A significant increase might indicate a failing T-cell response.

5.8 Reproductive and developmental effects

From the very few studies available no clear effects on reproductive health have been reported in humans. Animal studies have indicated adverse effects in rodents ingesting or being exposed via dermal contact to extremely high levels of soluble uranium compounds (WHO 2001).

Uranium has been shown to be present in the semen of veterans retaining fragments of DU shrapnel and presumably would be present in the semen of soldiers heavily exposed to DU aerosols. DU also appears in the testes of rats containing implants of DU pellets (Pellmar et al 1999a). This raises the possibility of adverse effects on the sperm from either the alpha-particles emanating from the DU or from the mutagenic activity of uranium, and

possible synergistic effects (Miller et al 1998a,b). Uranium is also known to cross the placenta (Sikov and Mahlum 1968; McClain et al 2001) and increased levels of uranium in the mother will lead to increased levels in the foetus.

Studies on the reproductive health of workers in the nuclear industry, and of survivors of the atomic bombs, show little evidence of decreased fertility, or an increased incidence of miscarriages or birth defects (Otake et al 1990; Doyle et al 2000). For example, a large study of over 20,000 pregnancies in the partners of male radiation workers at the Atomic Weapons Establishment, the Atomic Energy Authority and British Nuclear Fuels who had been exposed to radiation prior to conception showed no increase in foetal deaths or malformations. The lack of effect was seen both for workers who were only monitored for external radiation and for those monitored for both internal and external radiation. Female radiation workers exposed prior to conception had a slight increase in early miscarriages and stillbirths (Doyle et al 2000).

Effects of natural uranium on reproductive health have been observed in male mice, although at very high intakes. Daily ingestion of large amounts of soluble uranium (between 10 and 80 mg uranium per kg per day; equivalent to 700 mg - 5.6 g per day for a 70 kg man) over nine weeks had no apparent effect on testicular function or sperm development, but there were some effects on the morphology of the hormone-producing cells in the testes at the highest exposure level. A decrease in male fertility was reported but this was not related to the level of uranium exposure and its significance is unclear (Llobet et al 1991). We are not aware of any animal studies that have looked for developmental abnormalities in the progeny of uranium-exposed males.

In other studies using male mice injected with plutonium-239 and mated to untreated females, there was an increased susceptibility to leukaemia induced in the offspring by methyl-nitroso-urea (Lord et al 1998). The dose of plutonium (accumulated to three months prior to mating and averaged over the testis) which doubled the susceptibility to leukaemia in the offspring can be calculated to be around 100 mGy, ie about 2 Sv using the radiation weighting factor of 20. However, to achieve the same dose to the testes of a 70 kg man using the much less radioactive DU would require injection of about 1 kg of soluble DU.

Ingestion of 5 mg of soluble uranium per kg per day during pregnancy had no effect on sex ratios, mean litter size, body weight or body length of the newborn mice at birth or during the subsequent three weeks (Domingo et al 1989a). When treated males (ingestion of 25 mg uranium per kg per day for 60 days) were mated with treated females (25 mg uranium per kg per day for ten days prior to mating and subsequently), there were significant numbers of dead offspring per litter at birth and at day four of lactation. Also, the growth of the offspring was always significantly less for those derived from the uranium-treated animals (Paternain et al 1989).

Doses of 5-50 mg of soluble uranium per kg per day in food during pregnancy have been shown to reduce foetal body weight and body length, and to produce developmental defects including cleft palate and skeletal abnormalities (Domingo et al 1989b). These effects were particularly apparent at the 25 and 50 mg per kg dosages but some effects were apparent at 5 mg per kg. Developmental effects and malformations were also observed in mice born to mothers given daily subcutaneous injections that resulted in severe maternal toxic effects including death (Bosque et al 1993). The significance of these effects in mice are unclear as they occur at high intakes of soluble uranium that are the equivalent of between 250 mg and 2.5 g per day for a 50 kg (eight stone) woman.

There are uncertainties in extrapolating from animal studies to humans and there is a possibility of effects on reproductive health for soldiers who have high levels of exposure to radiation; careful epidemiological studies are required. Dr Pat Doyle and colleagues are investigating the reproductive health of male and female UK Gulf War veterans and the health of their children, although the results of the study are not yet available. The study compares those that served in the Gulf with a similar group of military personnel who were not deployed in the Gulf. The endpoints being examined include infertility, foetal loss, low birth weight, congenital malformation and childhood illness. If there is an effect on reproductive health, it will not be possible to establish whether this is due to DU or to any of the other potentially toxic exposures in the Gulf War.

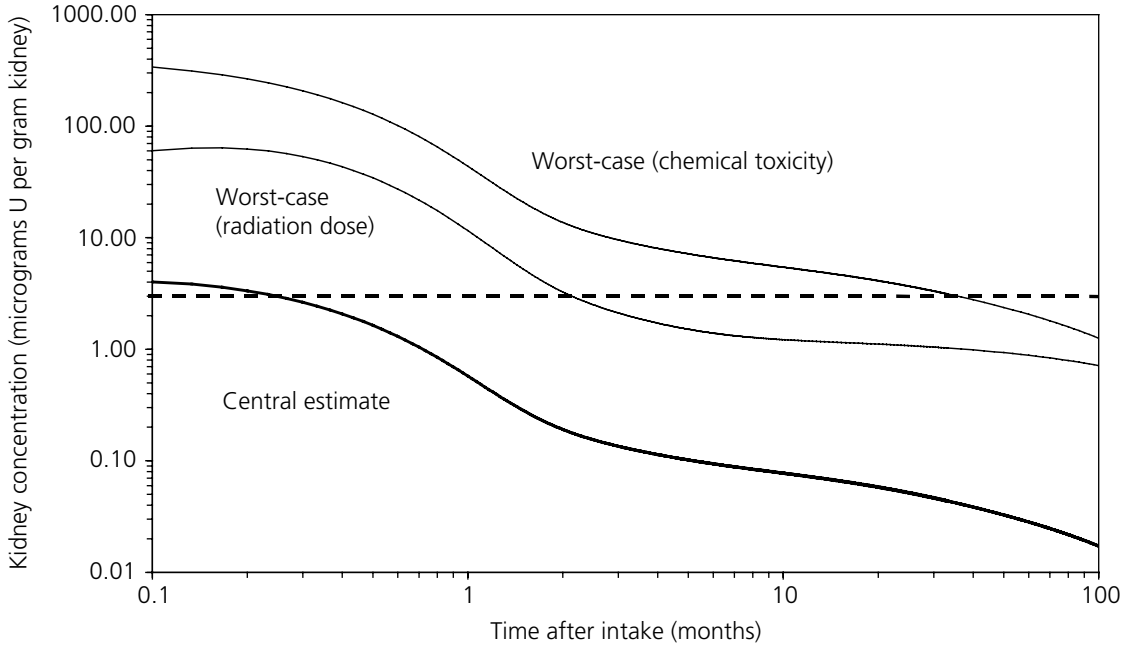
6.0 Kidney uranium levels and kidney effects from DU intakes on the battlefield

All of the available information indicates that the most serious adverse effects from the chemical toxicity of uranium will be on the kidney. In Part I of this report, biokinetic models were used to estimate the amounts of uranium reaching the kidney for the intakes of DU that might occur on the battlefield. Two estimates were obtained for each battlefield scenario. The 'central estimate' used the most likely values of the amounts of DU that could be inhaled (or ingested), and the most likely of the rates of dissolution of the inhaled or ingested DU. The 'worst-case estimate' used values of intakes of DU that are unlikely to be exceeded, and values of the dissolution rates of inhaled or ingested DU that maximise the amount that reaches the kidneys. The estimated maximum concentrations of uranium in the kidneys for different battlefield scenarios are given in table 7.

Table 7. Summary of predicted maximum concentrations of uranium in kidney (µg uranium per gram kidney) following DU intakes estimated for various scenarios. Values greater than or equal to 3 µg uranium per gram kidney are highlighted in bold as this level has often been used as the basis for occupational exposure limits. (From Part I, Appendix 1, table 27)

Scenario	Central estimate µg uranium per gram kidney	Worst case µg uranium per gram kidney
Level I inhalation of impact aerosol	**4**	**400**
Level II inhalation of resuspension aerosol within contaminated vehicle	0.05	**96**
Level II ingestion within contaminated vehicle	0.003	**3**
Level III inhalation of resuspension aerosol within contaminated vehicle	0.005	**10**
Level III ingestion within contaminated vehicle	0.0003	0.3
Level III inhalation of plume from impacts	0.0009	0.6
Level III inhalation of plume from fires	0.00012	0.05

Figure 4. Predicted concentration of uranium in the kidneys following an estimated Level I inhalation intake of DU oxide. Acute intakes of 250 mg (central estimate) or 5 g (worst case), and the parameter values from Part I, Appendix 1, table 14, are used. The levels of uranium in the kidney are shown for the central estimate and for the worst-case for chemical toxicity and for radiation dose; uranium levels are less under the conditions that maximise the radiation dose. The bold horizontal broken line indicates a concentration of 3 µg uranium per gram of kidney.

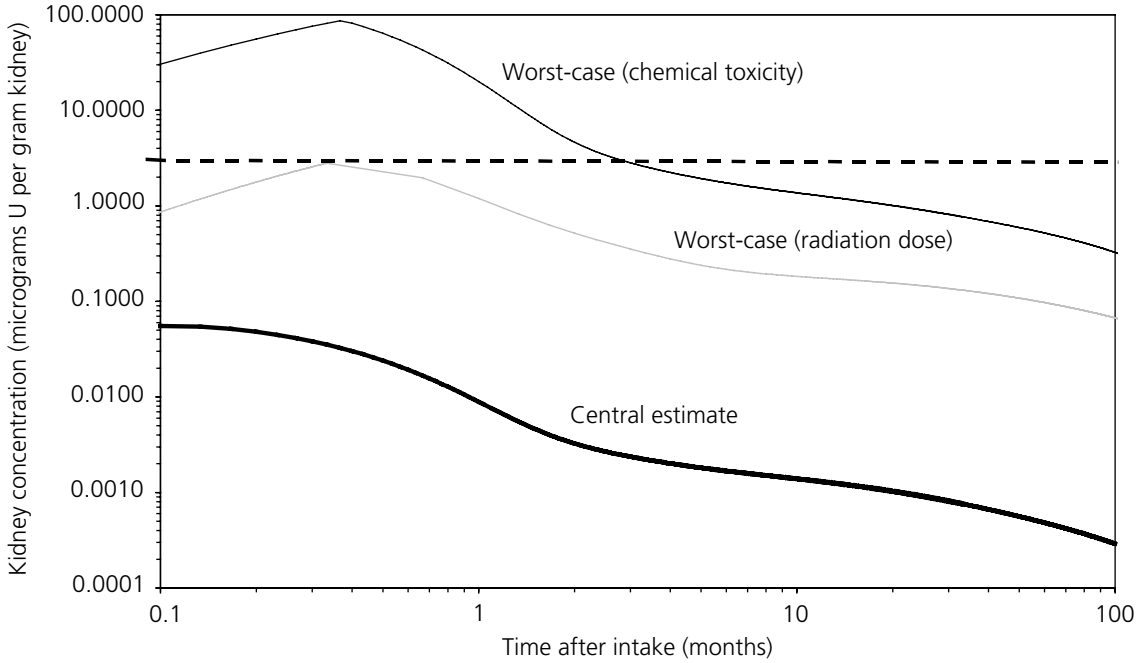


6.1 Kidney effects from central estimates of DU intakes

For the central estimates, the maximum concentrations of uranium in the kidney for the Level II ingestion scenario, and all Level III scenarios, are predicted to be ≤ 0.005 µg per gram kidney. It is improbable that these levels will lead to any significant effects on kidney function. The estimated maximum kidney concentration from the Level II inhalation exposure (0.05 µg per gram kidney) is slightly greater than the kidney uranium concentration in rabbits at chronic intakes that produced slight effects on the kidney (0.02-0.04 µg per gram kidney), and is about seven times greater than the kidney concentration estimated for the WHO tolerable daily intake. A kidney uranium concentration that transiently reaches a maximum of 0.05 µg uranium per gram is also unlikely to produce any long-term adverse effects on the kidney.

The central estimate for the Level I inhalation scenario predicts a peak kidney uranium concentration of about 4 µg per gram. From the limited information available on the toxicity of uranium in humans, it is considered that a concentration of 4 µg uranium per gram of kidney for

Figure 5. Predicted concentration of uranium in kidneys following an estimated Level II inhalation intake of DU oxide: acute intake of 10 mg (central estimate) or 2000 mg (worst-case) using parameter values from table 15 of Part I, Appendix 1. Note that the worst-case is based on 100 hours exposure at 20 mg intake per hour and is represented here by ten intakes of 200 mg on ten consecutive days. This results in a slightly lower maximum concentration (87 µg uranium per gram kidney) than a single intake of 2000 mg (96 µg uranium per gram kidney). The horizontal broken line indicates a concentration of 3 µg uranium per gram kidney.



about a week (Figure 4) is likely to cause some damage to the kidney. Kidney function can be reduced by as much as two-thirds without any obvious symptoms and soldiers exposed to DU intakes that transiently result in concentrations as high as 4 µg uranium per gram of kidney are unlikely to show any clinical signs of kidney dysfunction, although some dysfunction could well be apparent using biochemical markers of kidney function for a short period after the intake. Whether such an exposure would lead to any long-term effects or would increase the chance of kidney disease in later life is unknown, but we consider it unlikely.

6.2 Kidney effects from worst-case estimates of DU intakes

The worst-case peak concentration of uranium in the kidney arising from Level I inhalation exposures to DU is very high (about 400 µg uranium per gram kidney). This level greatly exceeds the occupational limit of 3 µg uranium per gram kidney, which is believed to be set at too high a level, and would result in uranium concentrations in the kidney above this occupational limit for a few years even supposing normal kidney function were maintained (figure 4). A very high peak kidney concentration (about 100 µg uranium per gram kidney) is also predicted for the worst-case Level II inhalation exposure and the level would remain above 3 µg per gram for several months (Figure 5).

The worst-case Level I and Level II inhalation estimates are greater than the peak kidney uranium concentrations predicted to have occurred in all of the cases of accidental exposure to uranium where very severe effects on the kidney were observed. It therefore seems likely that the worst-case estimates of the amounts of DU reaching the kidneys would lead to acute kidney failure that would be lethal in the absence of appropriate medical intervention. It is not clear whether our worst-case kidney uranium levels would actually occur after intakes of DU on the battlefield, as they assume the highest estimates of intakes for each scenario and the values of all of the parameters of the biokinetic models (aerosol size, solubility, etc) that maximise the amount of uranium reaching the kidney. If they did occur, they would be expected to apply to only a small number of those soldiers receiving Level I or II inhalation exposures, and should be very apparent, as they would be expected to result in acute distress and kidney failure soon after exposure.

The worst-case Level III inhalation scenario is also predicted to give a high peak kidney uranium concentration (10 µg per gram) and this level may also lead to some significant kidney damage. Peak concentrations of 3-4 µg per gram are estimated for the worst-case Level II ingestion and Level III inhalation from resuspension of DU from the ground.

Table 8. Summary of predicted maximum concentrations of uranium in the kidney following long-term DU intakes from resuspended soil

Scenario	Central estimate μg uranium per gram kidney	Worst case μg uranium per gram kidney
[1]Long-term inhalation of resuspension from ground:		
adult	0.002	0.2
10-year-old child	0.001	0.1
1-year-old child	0.001	0.1

[1]See Annexe F

6.3 Kidney effects from longer-term environmental DU contamination

Adults and children returning to live in areas where DU munitions were deployed will be chronically exposed to slightly elevated levels of uranium, by inhalation of DU particles from resuspended soil and by ingestion of contaminated food and water. The central estimates of the kidney uranium concentrations from these long-term inhalation exposures to DU are predicted to be less than the kidney uranium concentration at the WHO tolerable daily intake (table 8).

Worst-case estimates of the kidney uranium concentrations from long-term inhalation exposures for adults and children returning to areas where DU munitions were deployed are predicted to be 0.1-0.2 μg per gram. These chronic exposures would be expected to result in minor kidney dysfunction, as the kidney concentrations are greater than those where adverse effects were observed in the study of individuals chronically exposed to elevated levels of uranium from some private water sources (Limson Zamora et al 1998).

Intakes of uranium by ingestion from contaminated food and water, or by ingestion of soil, will be highly variable and are very difficult to estimate. There are no measurements that indicate any significantly elevated levels of uranium in Kosovo (although there are no data for Iraq) and attempts to estimate ingestion intakes, and resulting risks, have not been made, although they could be made if data became available through continued environmental monitoring.

6.4 Kidney effects from retained DU shrapnel

The excretion of uranium in some Gulf War veterans, some of whom had retained metal fragments and others who had not been exposed to DU, has been investigated (McDiarmid et al 1999).

A comparison of the amount of uranium in 24-hour urine collections and 'spot' urine collections was made. Results ranged from non-detectable to 30.7 μg uranium per gram creatinine. Where the uranium concentration was greater than 0.05 μg uranium per gram creatinine it was possible to use the 'spot' collection, but for lower amounts of urinary uranium, correcting for creatinine, concentration or volume did not give a satisfactory correlation with the 24-hour results. The authors concluded that for urinary levels below 0.05 μg uranium per gram creatinine, normally found in low-level exposed populations, 'spot' urine collections might be unreliable.

Thirty-three Gulf War veterans, 15 of whom had evidence of retained shrapnel on X-ray, were examined three and four years after the war. Measurements of uranium were made in 24-hour urine samples as well as 'spot' urine collections. The concentration of uranium was 150 times higher in those with X-ray evidence of shrapnel and the findings were similar a year later. The use of 'spot' urine collection was considered to be satisfactory (Hooper et al 1999).

Twenty-four-hour urine samples were collected from 169 Gulf War veterans between August 1998 and December 1999; urine uranium concentrations ranged from 0.001 to 0.432 μg uranium per gram creatinine with a mean of 0.02 and a median of 0.01 μg uranium per gram creatinine. These values were comparable to those of a non-DU exposed group of Gulf War veterans, assessed in 1997 (McDiarmid et al 2001). Reference ranges for a US population were determined by studying urine samples from a cohort of 500 people out of a group of 30,000. The mean was 0.01 μg uranium per gram creatinine, the median 0.006 μg uranium per gram creatinine and the 95th percentile value was 0.035 μg uranium per gram creatinine (Ting et al 1999).

As discussed in Section 4.2.5, ten years after the Gulf War these chronic exposures would be expected to result in about 0.5 μg uranium per gram kidney for the soldier with the highest uranium excretion level, and an average level of about 0.1 μg uranium per gram kidney. Such levels might be expected to result in some kidney dysfunction but no effects have so far been reported.

7.0 Conclusions

The chemical properties of DU are the same as those of enriched and naturally occurring uranium and it is therefore feasible to compare their toxicity. However, there are significant differences in the forms of DU and the modes of intakes of DU on the battlefield, compared with natural intakes of uranium and those that occur in industrial settings. The absorption, retention and excretion of uranium are dependent upon the chemical form and especially the solubility in biological fluids. The most common forms of uranium, following the firing of DU munitions, are likely to be the uranium oxides (UO_2, UO_3, and U_3O_8) and inhalation of these oxides presents the greatest risk of exposure. There is also a risk in war zones of shrapnel injuries with DU fragments.

The variability and uncertainties in the absorption rates of inhaled uranium oxides released in DU penetrator impacts or fires necessitate caution during the calculation and interpretation of uranium biokinetic data. For example, there is very limited information regarding the solubility and toxicity of ultrafine particles of DU. The behaviour in the body of uranium that is ingested is well understood. There are more uncertainties associated with the behaviour of inhaled uranium particles in humans, and in the absence of specific data on the solubility and bioavailability of the DU oxides (including the microfine component), extrapolation from the behaviour of uranium in animal models may not always be valid. More data are required on the dissolution and absorption characteristics of DU in the aerosols formed as a consequence of the combustion and thermal oxidation of DU that occur on the battlefield, and whether the inhalation toxicity of these materials in animals is different from that of other uranium oxides that have been studied.

Normal healthy adults may retain as much as 90 µg of uranium in the body from usual intakes of food and water. Uranium is retained principally in the kidneys and skeleton, or following inhalation in the respiratory system and associated lymph nodes. The critical organ for the biochemical toxicity is the kidney. The literature on uranium does not provide extensive evidence on chemically induced health effects in humans, mainly because there are few studies where substantial intakes of uranium have occurred in the absence of other confounding toxic intakes. A thorough quantitative risk assessment for the chemical toxicity of uranium in humans is difficult to achieve, as the information on exposure, both qualitatively and quantitatively, is inadequate. However, there is no clearly increased morbidity or frequency of end-stage renal disease in occupational populations chronically exposed to uranium concentrations above normal ambient levels. This is not necessarily reassuring, since the acute or short-term inhalation intakes of some soldiers on the battlefield are likely to be much greater than those that typically occur during chronic exposures in occupational settings.

Occupational studies are also restricted to the effects of uranium on healthy adults and provide no information on the more vulnerable members of the population such as children, the sick and the elderly. Children are not small adults and their exposure may differ from an adult in many ways. Children consume more calories per kilogram of body weight than adults and may have a higher gastrointestinal absorption of metals, possibly associated with higher lipid contents in their diets. In species like rats, in which skeletal growth occurs well into adulthood, uranium is continuously deposited in bones. Such deposition might occur in growing humans. However, few definitive data exist comparing paediatric and adult uranium exposures. Similarly, very little information is available on the inter-individual variation of uranium toxicity in humans. Kidney function deteriorates with age and reductions in kidney function resulting from toxic levels of uranium might be more serious in the elderly than the young.

Due to the paucity of data on the chemical toxicity of uranium to humans, most information is derived from animal studies. Although there is an extensive literature on animals, there is clear evidence of differences in response between species (Tracy et al 1992*).* The available data are fragmented, using a variety of animal models that differ in sensitivity to uranium (rabbits and dogs being more sensitive than rats by factors of two to ten). Only limited information is available on biokinetics or dose-effect relationships over a wide dose range. To some extent pharmacokinetic and metabolic processes are ignored, which makes direct interspecies extrapolation difficult. Inconsistencies can arise when comparing reports of measured kidney concentrations with reports in which kidney concentration has been estimated from intake. These factors add additional uncertainty in estimating a toxic threshold (Morris and Meinhold 1995).

The limited human studies suggest that damage to the kidney can be detected following chronic exposures that result in uranium concentrations as low as 0.1 µg per gram kidney. The human studies suggest that acute intakes which lead to peak uranium levels of about 1 µg per gram kidney can lead to detectable kidney dysfunction, and that those that lead to peak concentrations above about 50 µg per gram kidney may lead to kidney failure and death in the absence of appropriate medical intervention, although the latter value is based on a very small number of individuals exposed to such levels. It is likely that in single exposures or short-term exposures above the TDI, no adverse effects would be expected, but it is not possible to estimate with any confidence how long any exposures above the TDI could be tolerated, or how far above the TDI these exposures could be, without long-term adverse effects on the kidney.

Estimates of intakes of DU on the battlefield and of the concentrations of uranium in the kidney, under central estimate and worst-case assumptions, indicate that very high levels of kidney uranium could occur in a few soldiers under worst-case assumptions. For some soldiers who may have received high intakes of DU there is the possibility of slight adverse effects on the kidney even under central estimate assumptions.

Most of the effects of uranium have focused on its nephrotoxicity and there is very little information on other adverse effects of elevated levels of uranium in humans. There are studies that indicate some increased non-malignant respiratory disease in uranium workers but these are difficult to interpret. Although respiratory effects following a large inhalational intake would not be surprising, it is difficult to assess whether there would be long-term consequences. Effects on immune function are unlikely to be significant and would not be expected to lead to increased susceptibility to infection.

Those returning to live in an area where military action took place would be exposed to relatively low levels of uranium by inhalation and by ingestion. Although these intakes would increase the overall exposure to uranium, and may in some cases slightly increase kidney uranium concentrations, except in exceptional circumstances they would not be expected to be lead to any adverse effects on kidney function.

In laboratory animals exposed to low doses of uranium, functional abnormalities within the kidney are not detected until three to five days after exposure and may subside within seven days. Similarly, in humans acutely exposed to high levels of uranium, apparently normal kidney function was eventually regained, which may have implications for the monitoring and detection of adverse effects in humans.

Information on the monitoring and optimal treatment of the biochemical toxicity (as opposed to radiological risks) of uranium exposures is limited. There is no specific treatment for the chemical toxicity of uranium; treatment is symptomatic and supportive, aimed at supporting renal and respiratory function. A number of drugs (chelating agents) have been tested as methods to enhance the elimination of uranium, but the results have been disappointing.

Importantly, modern techniques are now available which are capable of detecting subclinical toxic effects on the kidneys, and in combination with the measurement of urinary (or plasma) uranium concentrations they should allow far more precise estimates of the risks of adverse effects from DU exposures.

In the lungs and associated lymph nodes of exposed individuals, and in soldiers with retained shrapnel, there will be high local concentrations of uranium around the retained DU particles or fragments. The possibility of synergistic effects from the damaging effects of alpha-particle traversals and the proposed direct mutagenic activity of uranium has been raised in Part I and needs to be considered further.

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Appendix 2: Depleted uranium—environmental issues

Barry Smith, Brenda Howard and Marshall Stoneham

1.0 Introduction

In military conflicts where depleted uranium (DU) munitions are deployed, soldiers may be exposed to a wide range of intakes of DU by a variety of routes. The consequences for health of these exposures have been considered in Part I of the report (radiological effects) and in Chapter 1 and Appendix 1 of this part of the report (toxic effects). The local population may also be exposed to DU during conflicts in populated areas, and there will also be long-term exposure to DU for civilians returning to areas where battles were fought, and for peace-keepers and aid workers. In addition to the deposition of particles of oxidised DU from aerosols produced during impacts of DU penetrators with their targets, there may be large numbers of minimally damaged penetrators on the ground or at various depths below the Earth's surface.

The corrosion of the large number of DU penetrators on and beneath the surface of the Earth can have environmental effects arising, for example, from uptake of the uranium by crops and grazing animals or from contamination of water sources.

In this appendix, we focus on the environmental behaviour of DU, the long-term consequences of its military use and the identification of areas where further research is needed.

1.1 Objectives

The overall objective of this appendix is to consider possible sources of DU in the environment and to estimate the magnitude of likely effects on ecosystems, groundwater and humans. Subsidiary objectives are to:

- define sources of DU in terms of their origin, and chemical and isotopic composition; consider the effect of the environment on the initial alteration and corrosion of DU
- review factors affecting the environmental transfer of uranium and DU, primarily focussing on pathways to man; review the environmental toxicity of uranium isotopes
- review factors influencing the contamination of groundwater by uranium isotopes
- identify key factors influencing human exposure to uranium isotopes
- consider the effects and likely impacts of DU use on ecosystems, groundwater and humans.

1.2 Uranium in the environment

Uranium (chemical symbol U) occurs naturally within the environment and is widely dispersed in the Earth's crust. Natural uranium is present to some extent in all rocks, waters and atmospheric particles. The abundance of uranium in the environment can be enhanced in several ways. Enrichment may happen where uranium minerals occur close to the soil surface and uranium becomes mixed with the overlying soil through weathering. Thus soils that have developed over uranium-rich rocks such as granites typically contain higher concentrations of uranium than soils developed over sedimentary rocks. Once released from uranium-bearing minerals and rocks into the environment, uranium may be dispersed resulting in an entirely natural plume (or halo).

Uranium concentrations in the environment can be enhanced by activities such as the mining of uranium and various other metalliferous ores (eg Ribera et al 1996; Burns and Finch 1999), emission from coal-fired power stations (eg NCRP 1975) and nuclear fuel manufacturing facilities (eg Efurd et al 1995; Meyer et al 1996; MAFF 1999; Ma et al 2000). Concentrations of uranium in a variety of materials and media associated with various potential exposure routes are highly variable and have been summarised in more detail in recent reviews (ie ATSDR 1999; WHO 2001). A brief summary is provided here.

1.2.1 Air

Background concentrations of uranium in air are present due to resuspended soil, and are typically low, ranging from less than 0.01 to 0.2 ng/m^3. Levels in air may be enhanced by the presence of various anthropogenic sources such as coal-fired power stations (eg NCRP, 1975), or facilities in which nuclear fuels are processed (eg Meyer et al 1996; MAFF 1999). Air concentrations can be also enhanced in the smoke from cigarettes (WHO 1998a).

1.2.2 Soils and sediments

The worldwide mean of the uranium content in different soils ranges from 0.79 to 11 mg/kg (Kabata-Pendias and Pendias 1984). Concentrations of uranium in soils and sediments in the UK vary widely and are typically 0.1 to 2 mg/kg in soils, and less than one to greater than 1000 mg/kg in sediments such as those that occur in stream and river alluvium (BGS 1974 to 2001; Regional Geochemical Atlas Series). The abundance of uranium depends upon its concentration in associated parent materials (ie rocks) or proximity of industries that may introduce uranium into the environment. Very high concentrations (up to around a factor of 100 times the typical ranges quoted above) may occur naturally.

1.2.3 Water

Concentrations of uranium in water are highly variable

Table 1. Typical range in $^{234}U/^{238}U$ activity ratios for various natural materials (as compiled by Ivanovich and Harmon (1982))

Material	Range in $^{234}U/^{238}U$ activity ratio
Open-ocean water	1.10-1.18
Terrestrial surface waters	0.80-2.50
Underground waters	0.60-12.00
Waters of uranium mineralisation	1.20-8.80
Various surficial carbonates	0.90-3.00
Fossil shells and bones	1.00-250
Peat deposits	0.90-2.00
Igneous rocks	0.60-2.10
Volcanic tuffs	0.50-1.60
Sandstones	0.80-2.00
Minerals and extracts of minerals	0.80-8.00
Soils	0.70-1.20

(typically 0.1 to 5 ppb). Observed concentrations depend upon: the concentration of uranium in soils and rocks within a given catchment; the proportion of surface water derived from groundwater; the solubility and mobility of the primary and secondary mineralisation and uranium speciation; and the presence of man-made sources (eg uranium mining or fuel-enrichment facilities). Uranium may also be introduced into the water by mining or various types of mineral extraction not necessarily associated with the commercial mining of uranium, in which uranium may be present as a component in other sources of mineralisation (eg coal or phosphate mining). Very high concentrations (up to around a factor of 100 times the typical ranges quoted above) may occur naturally in water and have been noted in many countries (Métivier and Roy 1998; WHO 2001).

1.2.4 Plants and animals

Measured uranium concentrations in vegetation and food crops range from 0.01 to greater than two micrograms per kilogram (compiled in WHO (2001)), whilst other estimates range from five to 40 microgram per kiolgram dry weight (Bowen 1979), although in making such measurements it is often difficult to exclude the possibility of sample contamination from the adhered dust of soil particles, particularly on foliage. Elevated concentrations of uranium may also be observed in plant species grown in contaminated environments (eg Rumble and Bjugstad 1986).

1.2.5 Diet

Typical total dietary intakes of uranium are in the order of one microgram per day, but most daily intakes within a country span an order of magnitude (UNSCEAR 2000). Of this intake, the major contributor is often tap or bottled water (WHO 1998b; ATSDR 1999). Other sources of baseline data related to the human intake of uranium through inhalation and ingestion include WHO (2001) and ATSDR (1999). Dietary intakes of uranium can be greatly enhanced by factors of over 100 in regions of high natural uranium abundance, especially where private water supplies are used (Finland - Kahlos and Asikainen (1980), Salonen (1988); Jordan - Gedeon et al (1994)).

1.2.6 Isotopic composition of natural uranium

The 'natural' isotopic ratio of $^{238}U/^{234}U$ is not constant in environmental materials. Variations (Table 1) may arise from a variety of environmental processes, which include the preferential leaching, and potential subsequent deposition, of ^{234}U due to crystal lattice damage resulting from the decay of ^{238}U to its daughter ^{234}Th, which then decays to ^{234}U (eg Fleischer 1983). The ratio of $^{238}U/^{235}U$, however, remains largely constant. A notable exception occurs at Oklo in Gabon where $^{238}U/^{235}U$ ratios have been influenced by natural nuclear fission (eg Burns and Finch 1999).

1.3 Legislation relating to the presence and use of uranium and DU in the UK

With a specific activity of 13-23 kBq/g, DU is a radioactive material within the meaning of the Radioactive Substances Act 1993. There are, however, a number of Exemption Orders under this Act that provide exemption from the need for registration of DU and/or authorisation for its disposal. These are reviewed in Jackson (2001) and include The Radioactive Substances (Uranium and Thorium) Exemption Order 1962, The Radioactive Substances (Prepared Uranium and Thorium Compounds) Exemption Order 1962, The Radioactive Substances (Waste Closed Systems) Exemption Order 1963 and the Radioactive Substances (Storage in Transit) Exemption Order 1962. The use of uranium and DU is also subject to the Ionising Radiations Regulations 1999 and various international safeguard requirements

established to control fissile materials.
From a purely chemical context, DU as a toxic metal is also considered in the special waste regulations 1996 and uranium is also defined as a List II Substance (EC Groundwater Directive, 80/68/EEC). This directive specifies that discharges of 'List II' substances into groundwater should be minimised in the UK and other member countries of the European Union.

The Environmental Protection Act 1990 also uses a number of definitions, which are relevant to the potential broader environmental impact of DU, covering issues other than the direct effect of contaminants on human health. For example, the definition of contamination as used to define contaminated land refers to the presence of hazardous substances in sufficient concentration to have the potential to cause harm (may be natural or man-made). In this act, a hazardous substance is defined as 'a substance with inherently dangerous quality' and harm means 'harm to health of living organisms or other interfaces with ecological systems of which they form part (in the case of humans includes harm to property)'.

There continues to be much debate regarding the level at which harm may be considered to be caused by the exposure of living organisms and ecological systems other than human beings. This is partly because of the potential breadth covered by such definitions. For example, soil may be seen as providing a wide number of functions in which any potential impact of a contaminant such as DU would have to be assessed to alleviate such concern. These functions include: the control of substance and energy cycles as a compartment of ecosystems; a basis for the life of plants, animals and humans; a carrier of a genetic reservoir; a basis for the production of agricultural products; and a buffer inhibiting movement of water, contaminants or other agents into groundwater.

1.4 Effects and risks associated with the release of DU

To determine the relative effects and risks of DU released during military conflicts on the environment, it is important to know not only the spatial variation in contamination by DU but also its origin and physicochemical form, and the extent to which different environmental factors affect its mobility and how these compare with exposures originating from the presence of natural uranium. The relative rates of transfer along different pathways will determine the importance of different routes by which various environmental receptors (ie groundwater, soil, ecosystems, etc) may become exposed. Furthermore, it is important to determine whether DU behaves similarly to natural uranium, and to identify the cause of any discrepancies and the implications for determining exposure for both. These factors and their likely effects on the environment are discussed in the following sections.

2.0 DU—source terms

Uranium is used as fuel in nuclear power plants and most reactors require fuel that is enriched in ^{235}U from its normal level of 0.72% to about 3%. DU is a by-product of this enrichment process and contains less ^{235}U (about 0.2%), and less ^{234}U, than natural uranium. Because DU contains less ^{235}U it is about 40% less radioactive than natural uranium. Theoretically there should be no significant differences in the chemical behaviour or toxicity of the different isotopes of uranium. Thus studies of the toxic effects of uranium as a poisonous metal can be directly applied to DU. However, any predicted radiological effects of natural uranium on health would be expected to be slightly less for the same mass of DU.

Natural uranium will also contribute to any toxic or radiological effects, although it may be present in forms which are less readily taken up (bioavailable) or leached into groundwater. In Section 2.1 we identify additional sources of uranium that arise from the various uses of DU. These will often be present in different chemical or physical forms to natural uranium. For a recent review of the occurrence and behaviour of natural uranium, see Burns and Finch (1999).

Uranium is chemically purified from ore as part of the nuclear fuel cycle and during this process the naturally present radioactive daughters of the uranium decay chain are removed. Therefore, purified uranium is much less radioactive than naturally occurring uranium ore, which still contains a significant number of high-activity daughter products. Once purified these natural daughter products of uranium begin to 'ingrow' into the purified uranium, resulting in an increase in the concentrations of ^{231}Pa, ^{234}Pa, ^{234}Th and ^{231m}Th. These ingrowing beta- and gamma-emitters are the main contributors to external dose, but their impact on internal dose is considered to be slight (see Part I of the report). Similarly, the presence of trace quantities of transuranic elements (eg plutonium and americium) and fission products (eg technetium) has been considered to be of little radiological significance (Royal Society 2001; WHO 2001). These elements are present at such low concentrations that toxic effects resulting from their purely chemical interaction with the human body are also expected to be limited.

In comparing the potential impacts of various sources of DU and/or natural uranium within the context of the natural environment, it is important to consider the relative spatial scale of both the source term and the potentially affected components of the environment. For example, an isolated point source of pollution for a large aquifer may represent a diffuse source to an individual agricultural smallholding. Thus, depending upon the size of the affected component of the environment, a military battle in which DU weapons

have been used may be considered as a single diffuse source of contamination or as a series of point-source contamination incidents. Such considerations place different demands on the selection of the most appropriate method to describe the source of contamination and the predictive models used to estimate the transfer of any contamination throughout the environment.

2.1 Potential sources of DU in the environment

Uranium has been mined and processed for use in nuclear reactors for several decades. DU is a by-product of the processing of natural uranium and it is plentiful and potentially cheap. Its high density makes it particularly useful for a range of commercial applications, which notably include radiation shielding, counterbalances and military hardware. Whilst uranium is naturally present in the environment, DU is not, and therefore the following discussions principally focus on the sources and characteristics of various forms of DU that may be released into the natural environment, rather than on the characteristics and forms of naturally occurring or enriched uranium that may be released as a result of mining and nuclear waste disposal.

2.1.1 Nuclear fuel cycle

Uranium is an essential component of the nuclear fuel cycle and as such may enter the environment in a wide variety of stages and isotopic compositions, from the initial mining of uranium ore to the recycling and subsequent disposal of nuclear waste. For example, facilities licensed by the appropriate national authorities in the UK for the release of uranium into the natural environment include mineral processing plants, enrichment plants and reprocessing facilities (eg MAFF 1999). The more radioactive isotopes of uranium such as ^{235}U and artificially produced ^{236}U are more stringently controlled and form part of a wide range of transuranic elements and fission products, which are intrinsic components of nuclear waste. Their release into the environment has therefore been extensively considered as one of the issues associated with nuclear waste disposal (eg Chapman and McKinley 1987).

On the other hand the by-product DU, which is less radioactive and cannot be used as the active component in nuclear weapons, is commonly stockpiled (often as UF_6). In terms of quantity, DU often constitutes the largest component of a country's nuclear inventory due to the low percentage of ^{235}U in natural uranium compared with that needed for nuclear fuel.

The current US stockpile of 'surplus' DU has been recently estimated to be between 500,000 and 700,000 metric tonnes (DOE 2000). This compares with a world output (at the mine in 1998) of 33,900 tons per year of uranium (BGS 2000) and a total estimated UK stockpile of around 60,000 tons of DU (Jackson 2001). Given the reactive nature of UF_6 the USA plans, as part of its ongoing clean-up programme, to convert its UF_6 reserves into metallic DU and mixed oxides of DU (DOE 2000). Potential uses for DU investigated during this exercise included aluminium refining electrodes, catalysts for fuel cells and steam reforming, catalysts for automotive exhausts, heavy vehicle counterweights, DU-based heavy concrete, oil well penetrators and drilling collars, package fill in nuclear waste repositories and conversion to uranium silicide for subsequent use in concrete.

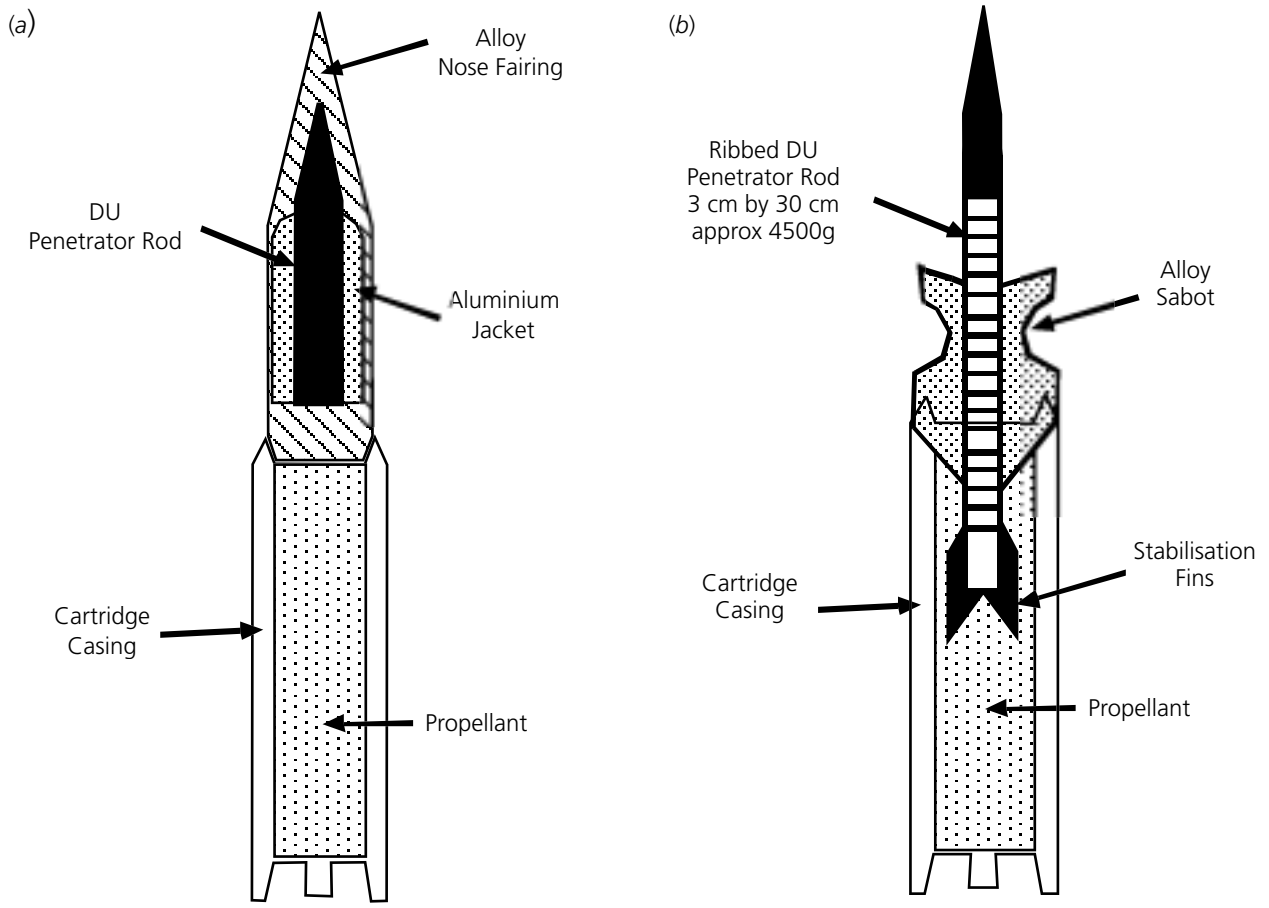
2.1.2 Aviation

Ingots of DU are present in some older aircraft and helicopters as counterweights (Jackson 2001). In aircraft the DU is either plated (cadmium and/or nickel) and painted or encased in a thin skin of aluminium alloy. In Part I of the Royal Society report the risks to human health from specific air accidents were not assessed, although the risks from aerosols of DU released in fires were considered. The people most likely to be exposed to DU are those working in the manufacture or service of counterweights containing DU. These exposures are likely to be significantly below the Level II scenarios developed in Part I of the report, and are estimated to be low, with minimal associated radiological risks, provided that adequate precautions such as those described in NUREG (1999) are in place. Exposure of other biota to DU from aircraft during routine service is limited to gamma- and beta-irradiation in the immediate vicinity of counterweights. The most likely scenarios in which more widespread environmental exposures to DU from aircraft could occur are those associated with an air crash, or when balance weights are inappropriately transported, stored or scrapped.

Records of the inventory of DU in different types of both civilian and military aircraft are often incomplete (Jackson 2001). The amount of DU used as a counterweight varies both between and within aircraft types and can change during an aircraft's lifetime during maintenance. For instance, if the DU corroded it could have been replaced by counterweights composed of a different material or a refurbished DU counterweight. This can make it difficult to define accurately both the source term and amount of DU released for any particular aircraft incident.

To date, the main focus of attention on the release of DU from aircraft has been on the consequences of the air crashes at Amsterdam and Stansted. Levels of DU introduced into the environment as a result of such air crashes depend on a wide variety of factors. Despite the potential difficulties outlined above, in the Stansted accident a large proportion of balance weights were reported to have been recovered in a near-intact condition with little signs of oxidation or damage having occurred (DETR Air Accidents Investigation Branch, Letter to RS EW/95/15, 21/10/00). In the case of the 1992 Amsterdam crash (a wide-bodied Boeing 747-

Figure 1. Schematic diagrams of: (a) 30 mm DU round (note the alloy fairing that covers the DU penetrator rod and associated jacket that remains with the penetrator until impact) and (b) 120 mm DU round (note that the alloy sabot that covers the DU penetrator rod is lost immediately after firing and the stabilisation fins are usually destroyed during impact).



258F), it has been reported in the press that only 130 kg of the initially estimated 282 kg of DU was recovered by clean-up teams and that the Dutch commission of enquiry concluded that some of the 'missing' DU may have been released in the form of oxide particles. Data presented in other recent studies such as Uijt de Haag et al (2000) confirm that approximately 152 kg of the DU from the crashed plane remained unaccounted for almost eight years after the crash. This does not necessarily mean that this quantity entered the local environment, as some or all of this material could have been removed from the site during general clean-up operations that included the removal of large quantities of topsoil (Uijt de Haag et al 2000). These issues illustrate the difficulties faced in assessing potential DU source terms associated with such accidents by either practical measurement or mathematical modelling.

2.1.3 Military hardware

DU is also used in the military sphere as a kinetic energy 'penetrator' in munition rounds designed to pierce the heavy armour of modern battle tanks. Such munitions are in the form of a long rod of DU. They carry no explosive charge but the large kinetic energy of motion of the very dense DU penetrator, travelling at speeds of up to 1.8 km per second, is sufficient to punch a hole in the armour of a modern battle tank. Unlike penetrators made of tungsten alloys, which blunt on impact with heavy armour, DU penetrators undergo self-sharpening on impact and have a superior penetrative ability. On impact with a hard target (such as a tank or other armour) the penetrator generates a cloud of DU dust within the struck vehicle that ignites spontaneously, creating a fire that increases the damage to the target. Sheets of DU, sandwiched between steel plates, are incorporated into the armour of some tanks, notably the heavy-armour variant of the Abrams M1A1 tank, and provide increased protection for the crew (eg AEPI 1995). Figure 1 schematically shows 30mm and 120mm DU rounds.

The first time it is believed that DU munitions were used in combat is the Gulf War of 1991, following concerns that tungsten penetrators might not be effective in destroying Soviet-built T72 Iraqi tanks. Currently DU munitions are used as armour-piercing rounds for the main armament of modern battle tanks (eg the British Challenger II and the American M1A1 Abrams); the 120 mm DU rounds typically fired by such tanks have

penetrator rods with DU masses of about four to five kilograms. During the Gulf War, approximately 9500 of these DU rounds were fired by US tanks and 88 by UK tanks (about 50 tons of DU) (Royal Society 2001), although other sources cited in AEPI (1995) suggest that more than 14,000 large calibre rounds were used by the US Army and Marine Corps alone.

Small calibre 30 mm DU rounds are used by the GAU-8 Gatling guns of US A-10 Warthog tank-busting aircraft. These rounds contain about 275 g of DU and an aluminium sabot surrounds the penetrator. They are typically fired in short bursts of about 100-200 rounds (typically a mix of one non-DU tracer round to every five DU rounds). It is estimated that about 780,000 of these 30 mm DU rounds (210 tons) were fired in the Gulf War (CHPPM 2000). Many of these may have missed their intended targets and may have penetrated some distance into the ground. Although large calibre DU rounds were not used in the Balkans, about 10,000 30 mm rounds (2.7 tons) were fired from US A-10 Warthog aircraft in Bosnia during 1994-95, and about 31,000 (8.4 tons) in Kosovo in 1999 (UNEP 2001; Royal Society 2001).

The total amount of DU in munitions fired during the Gulf War is subject to some uncertainty but has been estimated to be about 340 tons (CHPPM 2000), which is much greater than the approximately 11 tons used in the two Balkans conflicts. Neither Iraqi nor Serbian forces used DU munitions, although some anecdotal evidence suggests that NATO forces attacked a Serbian ammunitions factory containing DU rounds (N Priest, personal communication).

In addition to their use in combat, it has been a requirement of armaments use and development that a defined percentage of each round are test fired. In the UK proof firing of DU shells is performed at the MOD test site at Kirkcudbright (MoD 1995; Hansard written answers, 3 July 2001: Column: 96W). Developmental testing of the 120 mm DU ammunition has mainly been conducted at the MOD ranges at Kirkcudbright on the Solway Firth and Eskmeals in Cumbria, whilst testing of the Phalanx weapons system was performed at West Freugh (Luce Bay) but involved very small quantities of ammunition. A number of experimental kinetic energy DU rounds have also been fired within special contained facilities at Foulness. There have also been experimental tests of shaped charge anti-armour warheads containing DU liners at Aldermaston and Eskmeals. An environmental monitoring programme is operated by the MOD at Kirkcudbright, including the marine environment, and at Eskmeals (MOD 1995).

In the USA such testing has been performed at a number of sites including the Jefferson, Yuma and Aberdeen Proving Grounds (AEPI 1995), whilst in France testing of DU weapons has been reported to have been undertaken at Gramat (Barrillot 1994; ANDRA 1998).

2.1.4 Other uses

DU has been used to a limited extent in a wide variety of other applications including oil and gas exploration, civil engineering, shielding, dentistry, and as a colouring agent in the ceramics and glass industry.

2.2 The isotopic and chemical composition of the DU source term

2.2.1 Original composition

The exact chemical composition of DU depends upon source of manufacture and its end use. For example, uranium (or DU) may be alloyed with Ti, Mo or Nb (ASM 1991) and be incorporated into a complex metallurgical package.

DU as produced in the nuclear industry typically has around 0.2% to 0.3% ^{235}U by mass, although the Nuclear Regulatory Commission in the USA defines DU as uranium in which the percentage of ^{235}U is less than 0.711% (USNRC, 2000). Consequently, DU has a marginally higher percentage of ^{238}U (99.8%) than naturally occurring uranium (99.3%).

The ^{235}U content of DU in the US stockpile is considered to range from 0.2% to 0.711% (almost that of natural uranium) with an average of 0.27%, and 91% of the stockpile having a content of less than 0.4% ^{235}U (DOE 2000). The isotopic composition of DU typically used by the US Department of Defence in penetrators as quoted in CHPPM (2000) is ^{234}U = 0.0006%, ^{235}U = 0.2%, ^{236}U = 0.0003%, ^{238}U = 99.8% (all by mass).

During the development and testing of DU projectiles in the UK, the absence of gamma-emitting fission products was noted and the conclusion drawn that the DU used was essentially free from other radionuclides (MOD 1995). This has since been proven not to be the case, with a number of trace components being identified (eg Royal Society 2001; UNEP 2001).

In addition to being produced from mined uranium ore via the enrichment process, DU may also be obtained via the recycling of uranium irradiated in nuclear reactors. In some preparations of DU the material may therefore also contain transuranic elements and fission products (eg Rich 1988; CHPPM 2000). Such DU may consequently be very slightly more radioactive than DU derived from mined uranium ore. Typical trace isotopes identified as being present in DU used in munitions and armour manufacture by the USA and NATO include ^{238}Pu, ^{239}Pu, ^{240}Pu, ^{241}Am, ^{237}Np and ^{99}Tc. These impurities typically add less than 1% to the radiation dose from DU and are therefore inconsequential from a radiological or chemical toxicity standpoint. A recent survey of DU in Kosovo by the United National

Table 2. Chemical analysis of typical DU alloys (DU-Ti(0.75)) and uranium metal.

Element	DU mg/kg (wt%) Weirick and Douglass (1976)	DU mg/kg (wt%) Hasson et al (1981)	Uranium mg/kg (wt%) Kindlimann and Greene (1967)
Al	-	-	<1
Be	-	-	20
B	-	-	5
Cr	-	-	14
Ca	-	-	10
Mo	-	96	-
Nb	-	<10	-
V	-	<1	-
Al	-	35	-
Si	100	127	60
Fe	-	45	92
Cu	-	10	26
C	32	10	331
Ti	7450 (0.745%)	7100 (0.71%)	-
U	balance	balance	balance

—not tested for; < not detected.

Environment Programme provided a radiochemical analysis of penetrators found in conflict areas (UNEP 2001). The activity concentration of transuranic elements in these penetrators indicated that there was up to 12 Bq/kg of plutonium isotopes and for ^{236}U the activity was up to 61 kBq/kg. This compared with an activity concentration of 12,700 kBq/kg for ^{238}U.

The use of DU for military applications dates back at least to the early 1970s. US Navy, US Airforce and Canadians tested various types of munitions containing DU and associated alloys. In these early experiments, DU-2 wt% Mo alloy was the preferred alloy for such weapons, although the requirement for better corrosion resistance later led to the use of DU-0.75 wt% Ti alloys (Sandstrom 1976). DU as currently used in kinetic energy penetrator weapons is alloyed (at least in NATO arsenals) with 0.75% titanium, which significantly increases its strength and also its resistance to corrosion (Sandstrom 1976). The presence of other alloying elements of major or trace abundance has not been extensively documented or experimentally investigated in current studies such as those performed by UNEP (UNEP 2001). However, a brief summary of the concentration of non-radiogenic trace elements in DU and DU-Ti alloy based on available data is given in Table 2. The presence of such trace elements (eg carbon) may significantly affect the corrosion of penetrators and residual metallic fragments from such weapons and requires further investigation, as does the composition of DU used in kinetic energy penetrator weapons from other arsenals.

The chemical and isotopic composition of DU used in aircraft and other civilian uses as discussed in Section 2.1.4 above is not well described in the literature (eg Jackson 2001). Data requested from various manufacturers of equipment containing DU components (communication between Royal Society and manufacturers) suggest that in the vast majority of cases DU is used as an alloy (DU-Ti(0.75)) to reduce any potential effects due to corrosion and for purely logistical reasons (DU-Ti being the most commonly produced alloy).

2.2.2 Composition upon release into the natural environment

The chemical and mineralogical forms of DU introduced into the natural environment are difficult to quantitatively characterise for every potential scenario. The isotope ratios and the trace element compositions will both vary. For example, in the case of military uses the chemistry and relative proportion of discharged DU will be heavily dependent upon the nature of the penetrator impact (ie type and composition of penetrator, energy of impact, composition of impacted material) and of any subsequent chemical alterations occurring when debris interacts with soil or water.

2.2.2.1 Air crashes and other anthropogenic sources

The forms and composition of DU released during air crashes are likely to be broadly similar to those released during military conflict, particularly as evidence suggests that DU alloys used in such situations are similar to those used in military equipment (DU-Ti(0.75)). Experience from crashes at Amsterdam and Stansted suggests that

the majority of DU components remain in a relatively intact condition, hence reducing the potential for combustion. Where combustion of DU takes place it is likely that this occurs at a much slower rate than that occurring during the impact of armour penetrating munitions, and that mixed oxides of DU in the form of UO_2 and U_3O_8 are formed (eg Totemeier 1995; Parker 1988, Uijt de Haag et al 2000). Similar compositions of dust are likely to result from fires involving DU, for example the Camp Doha fire in the Gulf Conflict (eg CHPPM 2000) and those occurring at the Featherstone armaments factory (see Part I of the report, Annexe H).

DU produced as a by-product by the nuclear industry is commonly stored in pressurised containers as UF_6. Uranium hexafluoride is a highly volatile solid that within a reasonable range of temperatures and pressures may be a solid, liquid or gas. On mixing with moist air UF_6 rapidly hydrolyses to form hydrogen fluoride (HF) and UO_2F_2.

In the UK chemical plants associated with the isotopic enrichment of uranium have discharge limits, which are licensed through the relevant governmental agency. For example, discharge limits set for the BNFL Springfields fuel production facility during 1999 were 0.006 TBq (237 kg assuming a natural isotopic composition) for gases and 0.15 TBq (5933 kg assuming a natural isotopic composition) for liquids (RIFE 2000). These discharge limits are based on the total quantity of various uranium isotopes discharged rather than on any specific chemical and/or physical form of uranium. Uranium discharged from such sites becomes incorporated into the local natural environment and has been observed in soils, grass, stream sediments and tree bark (BGS 1999; RIFE 2000; Ma et al 2000). The isotopic composition of material discharged at these sites is variable. However, on average, data indicate release of enriched uranium and ^{236}U rather than DU.

2.2.2.2Military conflict

The nature and quantity of discharged DU has been reasonably well characterised during testing and on firing ranges (Royal Society 2001). However, there are few data or studies that allow a comparison to be made between the composition and form of discharged DU under controlled conditions and those during a military conflict. Similarly, because of the recent development of such munitions, there are virtually no data or studies that describe changes in the composition and form of discharged DU munitions over environmentally significant timescales. For example those in excess of ten years and more probably greater than 50 years or longer, which reflect periods over which uranium is likely to undergo translocation and mixing with surface soils and groundwaters. The environmental context may therefore involve periods much longer than human lives and contaminated land may be a concern for hundreds of years.

Tests conducted by the US ballistics research laboratory have shown that, although DU particles thrown into the air can travel downwind, the largest amounts of DU dusts created on impact come to rest inside a penetrated vehicle, with significant amounts on the outside surface and within ten metres of the target (SAIC 1990). Further information, citing tests on hard targets at the Nellis Air Force Range in the USA, indicated that DU dust from the impact of a 30 mm munition strike was deposited within 100 m of the target. Similar tests against a hard target with 120 mm DU munitions resulted in 90% (ie 4365 g out of a total mass of 4850 g) of the DU residue being deposited within about 50 m of the target (CHPPM 2000). Such dispersal patterns remained typical even after a fire began in a test tank and continued for in excess of 12 hours (AEPI 1995). In test firings on the Kirkcudbright range, there is evidence that more DU (concentrations of up to 240 Bq/kg in soil) was found nearer the guns (at Balig and Doon Hill) than near the targets (eg terrestrial data for August 1997 (Armstrong 1999)). This was presumably due to break-up of penetrators during firing.

For the purposes of this report, the composition of source term material has been characterised by considering two groups.

(1) *Uranium-rich dusts generated during impact and subsequent fires.* The compositions of dusts generated by impacts of DU penetrators have been classified according to their particle size distribution, major element chemical composition and solubility in synthetic lung fluids. These are discussed and described in detail in the appendices and annexes of Part I of the report and are summarised in Table 3.

    A review of experimental studies undertaken on the oxidation of pure uranium in oxygen and dry air by Totemeier (1995) indicates the formation of superstoichiometric UO_2 (UO_{2+x} where x = 0.2 to 0.4) below temperatures of about 300 °C and U_3O_8 above 300 °C.

    Recently published studies on samples collected from Kosovo by UNEP and the UK MOD illustrate the interdependency of both the physical and chemical form of particulate DU materials on the nature and type of material impacted upon. For example, a number of uranium-rich particles of between one and ten microns, with a highly variable chemical composition, containing uranium, calcium, silicon, aluminium and oxygen, with minor amounts of iron and titanium, were detected in the vicinity of a strafed compound that included a block-built concrete building (Milodowski 2001). This was despite little evidence of weight loss due to combustion of DU from the penetrators that struck these buildings (MOD 2001).

Table 3. Summary of the chemical and physical properties of uranium-rich dusts generated during the impact of DU penetrators and subsequent fires

Property	Description
Chemical composition	18%-60% UO_2, 40%-75% U_3O_8
Particle size distribution: close to impact	
Mass median aerodynamic diameter[1]	~2 microns
Geometric standard deviation[1]	~10
Particle size distribution: distant from impact	
Mass median aerodynamic diamete[1]	~1 microns
Geometric standard deviation[1]	~2.5
Solubility/absorption characteristics in biological media	10%-50% considered to be rapidly dissolved in lung fluid (*in vitro* tests only)

[1]see Annexe A, Section A2.3, of Part I of the report for definitions.

(2) *Residual metallic fragments and nearly intact penetrators.* Residual metallic fragments and nearly intact penetrators will have a bulk composition similar to those described in Section 2.2.1, with the exception that stresses during firing and subsequent impact may have caused micro-structural changes in the metallic penetrators. Such stress cracks and evidence of the formation of U(VI) corrosion products in 30 mm DU penetrators from Kosovo have been observed and reported (UNEP 2001).

In the case of small calibre munitions, such as those fired by A-10 aircraft, nearly intact penetrators have been found with the aluminium fairing still attached (UNEP 2001). In such cases, the presence of the aluminium fairing could significantly reduce the corrosion rate of the associated DU penetrator. It is not known how often the fairing and penetrator remain intact, although this may be more likely on impact with soft targets such as soil.

2.3 Environmental context

Perhaps the major factor that determines the environmental fate of DU is the location of the source term within the environment. In an extreme case, contamination of a well used for drinking water is inherently more likely if the penetrator directly enters the well than if it enters the soil in an adjacent field. It is also important in considering the environmental context of uranium and DU derived from man-made sources to consider time periods much longer than those associated with immediate post-conflict assessments. For example, the presence of DU in groundwaters used for drinking water may not be evident for many decades or lifetimes. As such, land contaminated with significant quantities of DU may be of concern for many hundreds of years. These factors are discussed below with reference to various scenarios and associated sources. The quantity, form and spatial distribution of discharged DU released into the environment following military activities are related to the type of military action and the consequent density of munitions use. These factors also influence the proportion of residual metallic DU (close to 100 % uranium metal), and aerosols and dusts containing mixed oxides of DU released into the environment.

2.3.1 Uranium-rich dusts

Dusts consisting predominately of mixed DU oxides and other components associated with energetic impacts or weathering reactions (eg calcium, carbonate, aluminium, iron, silicon, etc) may be generated during the impact of penetrators and subsequent fires, and/or through the burning of DU-based materials. Therefore the production of dusts must be considered in all military actions, including the testing of DU rounds and where fires have occurred. Dust production would be expected to be greatest where DU rounds directly hit armoured targets. Preliminary data available from the Kosovo conflict suggest that dust production may be minimal during impacts between penetrators and concrete structures (MOD 2001).

Further, recent data provided by UNEP (UNEP 2001) and other third parties (reported at a recent IAEA workshop on DU; IAEA Training Workshop, DU, Vienna 2001, which included representatives from Kosovo, Serbia, Iraq, Kuwait and Macedonia) suggest that most of the DU entering the environment following the use of 30 mm munitions appeared to remain close (generally within one meter) to an individual penetrator strike. On a broader scale, dispersed contamination was noted to be measurable for up to 50 m from an impact site (UNEP 2001). This is perhaps unsurprising given that typical strafing attacks probably resulted in over 100 such penetrator impacts in an area of around 1000 m^2.

Parallel studies reported by UNEP (2001), in which moss and other biological materials were analysed for DU, indicate that some atmospheric dispersal may have transported DU into areas where direct ground contamination from penetrator sites was absent.

Further research to establish the chemical and physical form of dispersed DU following the use of DU munitions in actual military conflict is currently being undertaken.

The two major factors that control the environmental context of these uranium-rich dusts are the force of impact and the composition of impacted material.

2.3.1.1 Force of impact
As a result of the high temperatures that are created during impact with a heavily armoured vehicle, uranium may be converted to a series of oxides, which include the relatively insoluble triuranium octaoxide (U_3O_8) and uranium dioxide (UO_2) (CHPPM 2000). Subsequent reaction of these oxides with atmospheric oxygen, water and CO_2 will typically produce relatively soluble uranium trioxide (UO_3) and associated U(VI) complexes (see the following section on corrosion). It has been stated that the relative insolubility of some of these oxides delays the rapid infiltration of dissolved uranium through the soil zone and into groundwater reserves. However, it does not preclude the physical migration and contamination of surface water resources with particulate uranium, or conversion into more, or less, soluble forms through interaction with other components of the target or soil.

Estimates of the quantity, solubility and particle size distribution of dusts produced during the discharge of DU weapons and in fires vary considerably because of the wide variety of potential impacts under combat conditions and the experimental limitations (CHPPM 2000; WHO 2001; Royal Society 2001). For example, AEPI (1995) cite studies indicating that up to 70% of the DU in a given projectile may be converted to dusts and aerosols on impact. Other more recent reviews (CHPPM 2000) cite lower estimates of 10% to 37%, for a range of hard target perforations.

2.3.1.2 Composition of impacted material
The chemical composition and crystalline structure of particles and aerosols produced during the impact of DU projectiles also depend upon the composition of the target material. The morphology and exact chemical composition of each particle released during the use of penetrators and armour are highly variable (eg Patrick and Cornette 1977). For example, studies by Patrick and Cornette (1977), and summary text from CHPPM (2000), indicate that complex spherical particles rich in DU, iron and titanium can be produced through high-velocity collisions with armour. The same authors also state that similarly shaped, complex particles may be formed by fusion with clay and sand (ie containing aluminium, potassium, silicon) as a result of direct impacts with soil or when hot, reactive, secondary particles from the initial impact interact with the soil environment. Moreover, they may be chemically and mineralogically altered by weathering either following the impact with the target or during their initial release into the environment (eg uranium oxides may become hydrated, chemically reacting with other elements and species present in the soil, and/or the struck target, such as aluminium, silica, iron, phosphate and vanadium (Patrick and Cornette 1977; Ebinger et al 1990)).

2.3.2 Residual metallic fragments and penetrators

2.3.2.1 Tanks
AEPI (1995) summarises experiences relating to the use of 120 mm armour-piercing munitions during the Gulf conflict. Of particular relevance to the environmental context are observations that DU penetrators from these munitions commonly passed completely through an armoured vehicle and that tank commanders often fired more than one DU round as the initial hit did not cause the target to explode. Estimated hit rates were in the order of 80% to 90%. In the 10% to 20% of cases where penetrators missed their intended targets, they were considered to be capable of ricocheting and skipping across the ground for in excess of one to three kilometres (AEPI, 1995).

Dusts containing mixed DU oxides commonly contaminated hit vehicles and, whilst enemy vehicles were generally left in place, allied vehicles were recovered and decontaminated prior to shipment or burial (AEPI 1995; CHPPM 2000).

During clean-up operations following the Gulf Conflict, nearly intact penetrators from 120 mm rounds and associated fragments were often found on the ground surface. In this context it must be understood that the Gulf Conflict occurred over an area of desert terrain in which bare rock and calcrete (a hard surface crust formed under desert conditions) were perhaps more common than deep sand. Over 500 DU penetrators of unspecified type were handed in following the conflict.

Additionally, AEPI (1995) estimates that it is possible for over half of the 120 mm DU rounds used by the US Army and Marine Corps to have been fired into large sand mounds in Saudi Arabia for practice and validation of fire control systems. The fate of penetrators fired into these mounds is not reported in AEPI (1995), although it is likely that penetration into the mounds would have been substantially in excess of that reported by UNEP (2001) for 30 mm munitions (zero to seven metres) due to the higher kinetic momentum of the 120 mm rounds.

2.3.2.2 Strafing
In US airforce tests prior to the Gulf War, a 'typical' A10 Thunderbolt strafing attack scenario against a T-62 tank resulted in a 90% miss and 10% hit rate (CHPPM 2000). This indicates that a substantial mass of DU might become buried in a rural environment and lead to subsequent dispersion in the soil and leaching into groundwater as a result of chemical weathering.

The depth to which DU projectiles penetrate into soil depends on the mechanical and physical properties of the soil profile. However, information on the relationship between penetration depth and soil characteristics has not yet been reported in the open literature. This uncertainty coupled with difficulties in identifying DU penetrators that have missed their target and become embedded in the soil profile represent a significant knowledge gap, particularly where targets have been strafed and the proportion of penetrators hitting a hard target is low.

In some cases in the Gulf War DU projectiles went through the target without oxidising or producing significant quantities of dust and aerosols, resulting in relatively large pieces of metallic DU entering the environment. In Kosovo it is considered that projectiles impacting into soft soil may penetrate into the ground to a depth of up to seven metres with minimal production of DU dusts (UNEP 2001).
The percentage of such buried projectiles depends on engagement angles, ranges and terrain (AEPI 1995) and is therefore variable. Little firm quantitative survey data appear to have been published on the potential depth penetration of projectiles into soils beyond observations that intact 30 mm and 25 mm penetrators have been found at a depth of 30 cm in soft soils typical of the Gulf or Serbia (CHPPM 2000; UNEP 2001). This is presumably because of the difficulty of detecting the beta- or gamma-radiation from buried DU projectiles. Projectiles that miss the target may also ricochet, skipping across the ground with minimal production of dusts and aerosols. During firing and impact the DU alloy in penetrators is subject to a wide range of physical stresses as a result of the intense forces produced during acceleration and impact. As discussed earlier metallurgical changes associated with these stresses, such as the production of micro-fractures, are likely to exhibit a profound effect on any subsequent corrosion of penetrators and consequently the MoD have proposed to undertake corrosion studies on both fired and unfired penetrators (R Brown; MoD personal communication).

2.3.2.3 Fire
Fires potentially involving the ignition and dispersal of DU have occurred at sites manufacturing or storing DU munitions (eg at Featherstone in the UK and at the Camp Doha ammunition dump during the Gulf War) and where tanks containing DU munitions have caught fire.

Like many metals Uranium, and hence DU, is pyrophoric in air. Parameters used to describe pyrophoricity include the ignition temperature (ie the temperature at which heat production from the oxidation process exceeds that of the local environment) and the burning temperature (ie the temperature reached during combustion). Experimentally derived data for 8.5 mm cubes of pure uranium as cited by Totemeier (1995) indicate an ignition temperature of around 600 °C and a burning temperature of approximately 1300 °C in an atmosphere of 20% O_2 / 80% N_2. However, in the same review Totemeier (1995) also cites data indicating that ignition temperatures may be:

(a) raised or lowered by a factor of approximately 10% depending upon the alloying of uranium
(b) lowered to around 300 °C when uranium is present as a fine powder, due to the effect of high specific surface area.

Totemeier (1995) cited a number of studies indicating the importance of using the ignition temperature of the finest sized particles (highest specific surface area) when estimating the ignition temperature of an aggregate of different sized particles. The heat generated by ignition of the finer particles was sufficient to heat the larger particles to their ignition temperatures.

Elder and Tinkle (1980) have investigated the effects of simulated fires involving penetrators in storage or during transport. Experiments involved the initiation of semi-controlled conditions exposing the penetrators to high temperatures, an oxidising atmosphere and an intermediate wind speed of 2.23 m/s (five miles per hour). It was observed that penetrators did not tend towards self-sustained burning; this only occurs when finely divided uranium is oxidised. Depleted uranium aerosols were found to disperse in all forced draft oxidation experiments at temperatures in the range 500 to1000 °C. In an outdoor burning experiment with temperatures up to 1100 °C, 42% to 47% of the penetrator by weight was oxidised in a three hour burn. Outdoor burning also produced greater quantities of aerosols in the respirable range (less than ten microns AMAD), with 62% of aerosol mass being in this size range compared with a maximum of 14% in the laboratory experiments. In general, DU aerosols in the respirable range are produced when penetrators are exposed to temperatures greater than 500 °C for burn times of longer than 30 minutes.

Other burn tests performed on DU munitions cited in CHPPM (2000) by Hooker et al (1983) and Haggard et al (1986) indicate that up to 90% of the DU may be oxidised under extreme conditions (eg two days within an active fire).

In the fire at Camp Doha, CHPPM (2000) estimated that 3090 kg of DU formed the source term of metal available for oxidation and dispersion. The majority of this material was present in ammunition (penetrator and propellant in cartridge case) stored in MILVANS trailers and conexes (storage containers). However, the source term also included munitions stored in three tanks. One major concern was therefore that at the high temperatures involved, ignition of the propellant would lead to significantly increased environmental dispersion

of DU. This was found not to be the case. Based on observations following the fire at Camp Doha, it was concluded that less than 15% of the total DU stockpile was oxidised and therefore potentially present as a contaminative dust (CHPPM 2000). Modelled airborne dispersion from the fire at Camp Doha suggested that dispersion occurred over a distance of up to two kilometers from the point of origin. Unfortunately, no site-specific data appear to have been collected on the particle size distribution and chemical form of uranium produced by the fire, extrapolations being made from controlled experiments performed during the 1980s and 1990s on the oxidation of DU during projectile fires. Based on these assumptions, modelled radiological doses and chemical doses derived from inhalation exposures were considered to be low (CHPPM 2000).

2.3.2.4 Proving and testing
Proof testing and developmental testing of DU munitions have been performed for at least 30 years (eg AEPI 1995; MOD 1995). As concerns over the environmental acceptability of DU munitions have grown, an increasing degree of sophistication has been used at testing sites to:

(a) characterise the type and quantity of sources of DU contamination that may result from actual impacts
(b) to proof test munitions
(c) to establish and verify targeting data (ie flight trajectories) and
(d) to test and demonstrate the effectiveness of integrated armament systems.

This sophistication has included the development of protected areas in which DU penetrators may be fired at armoured targets, for example the enclosed 'superbox' facility at Ford's Farm in the USA. Prior to 1980 virtually all of the activities described above were undertaken on open ranges, particularly at Aberdeen and Yuma Proving Grounds in the USA (eg AEPI 1995).
In studies undertaken to investigate scientifically the mechanics of impact, the collection of impacted materials is generally required; this type of material is usually obtained by positioning a 'catch box' of sand behind the target area (AEPI 1995; MOD 1995). In the UK this technique has been utilised at the Eskmeals range since 1981, using the VJ Butt enclosed sand 'Butt' which has been developed to offer near-complete enclosure of the target material. It has been estimated that about 350 test firings have been undertaken at VJ Butt since 1981 (MOD 1995). Sands from such 'catch boxes' are collected and appropriately disposed of under guidance from local regulatory bodies (depending upon levels and national regulations, such materials may or may not be classified as nuclear waste). At Kirkcudbright, the primary objective is to proof test DU munitions and to test the behaviour and accuracy of the trajectory of individual projectile configurations. Such firing is intended to be non-destructive, being aimed at soft targets through which projectiles pass before ending up in the sea (MOD 1995). Whilst this has the advantage of minimising the production of dusts and contaminated wind-blown material, it suffers the obvious disadvantage of introducing DU into the marine environment. Some of these projectiles have ended up impacting on the land, due to unpredicted changes in trajectory, where they either partially disintegrate on impact or become buried in soil. During this testing there are inherently also occasions during which the penetrator may fragment prior to, or immediately after, exit from the gun barrel. Under such circumstances contamination of the environment immediately surrounding the gun also occurs. Since 1982 the MOD have estimated that over 4000 DU rounds of various weights and designs have been fired into the sea off the Kirkcudbright range, where the vast majority remain in an unknown condition at unknown locations.

Armament systems using DU-based weapons have also been tested during training and during studies on their effectiveness under simulated battlefield conditions. For example, such testing occurred at the Aberdeen Proving Grounds in the USA from the 1950s until 1979, when the US NRC prohibited destructive testing that released airborne radioactive material to unrestricted areas. Under such circumstances areas of land at Aberdeen Proving Grounds became grossly contaminated, although not necessarily to a harmful extent, with DU. Whilst such sites as Aberdeen or others such as Yuma and/or Jefferson Proving Grounds may be used to study the environmental dispersion of DU, a number of factors such as the density of DU use compared with use in actual conflict situations, and the absence of a human population in such training areas, hinder extrapolation.

3.0 DU–corrosion and weathering of discharge products

A wide range of investigations have centred on the environmental behaviour (eg corrosion and transport) of uranium as various forms of oxide derived from the nuclear industry (eg high- and low-level nuclear wastes, etc). In the course of these investigations, experimental studies in the laboratory and those performed in the field have established that natural uraninites and their alteration products can be used as natural analogues to study the corrosion of UO2 in spent nuclear fuel. However, complementary studies have not been performed to indicate if they may also be used for the corrosion and subsequent transport of DU used in penetrators. Where possible, the data presented in the following sections therefore compare and contrast information related to the behaviour of both pure uranium metal and DU alloys as used in kinetic energy penetrators.

3.1 Corrosion
Corrosion is the general name given to a wide range of complex physical and chemical processes that result in

detrimental changes to the fabric and structure of a given metal. Corrosion is analogous in many ways to natural weathering processes, in which the breakdown or decay of rock on the surface produces a mantle of waste that may be subsequently eroded or transported. After firing, a penetrator may interact with the intended target or local environment producing either metallic fragments of DU alloy, or dusts and aerosols containing oxidised reaction products (eg UO_2 and U_3O_8). The corrosion of residual alloy penetrators or fragments entails oxidation of zero-valent metallic uranium to U(IV) followed by oxidation of U(IV) to U(VI) under favourable conditions. In contrast, the particulate material formed during impact with hard targets entails only oxidation of U(IV) to U(VI), again under favourable conditions (note that in some cases it may be possible for oxides of a higher oxidation state than U(IV) such as U_3O_8 or UO_3 to be directly produced during the impact event). In either case, corrosion in environmental matrices, and hence under environmental conditions, may be viewed as a chemical reaction between a series of defined materials and their local environment (eg chemical weathering), and the subsequent transport of reaction products away from the reaction site, thereby allowing continued exposure of fresh material. These are discussed in the following sections.

3.2 Corrosion of metallic DU

In the natural environment, metallic uranium or DU and associated alloys may corrode through a number of processes (eg galvanic corrosion, crevice corrosion, pitting corrosion), the majority of which are controlled by the local chemical environment surrounding the metallic uranium or uranium alloy. For example, corrosion may occur in air, water, or in contact with the water- and air-filled pores of soils and sediments. In addition to understanding the pure thermodynamics and kinetics of corrosion reactions, it is essential to consider the removal (or mass transport) of reaction products. These secondary phases, such as oxides, may be less reactive than the initial pure metal phases, forming a 'passive' barrier through which both reactant and reaction product must pass.

Experimentally, three factors have been observed to control corrosion processes and rates of DU under environmental conditions (Annexe G):

(a) the physical form of DU (ie surface area available for reaction, microstructure, crystal structure of fired DU)
(b) the chemical composition of the DU which is in contact with the environment (ie nature of the alloy, composition of DU, etc)
(c) the chemical composition and physical state of environmental reactants with which the uranium metal may be in contact (ie fluid or gas composition, local Eh conditions, soil chemistry, etc).

These factors are discussed in the following sections.

3.2.1 Processes of metallic DU corrosion

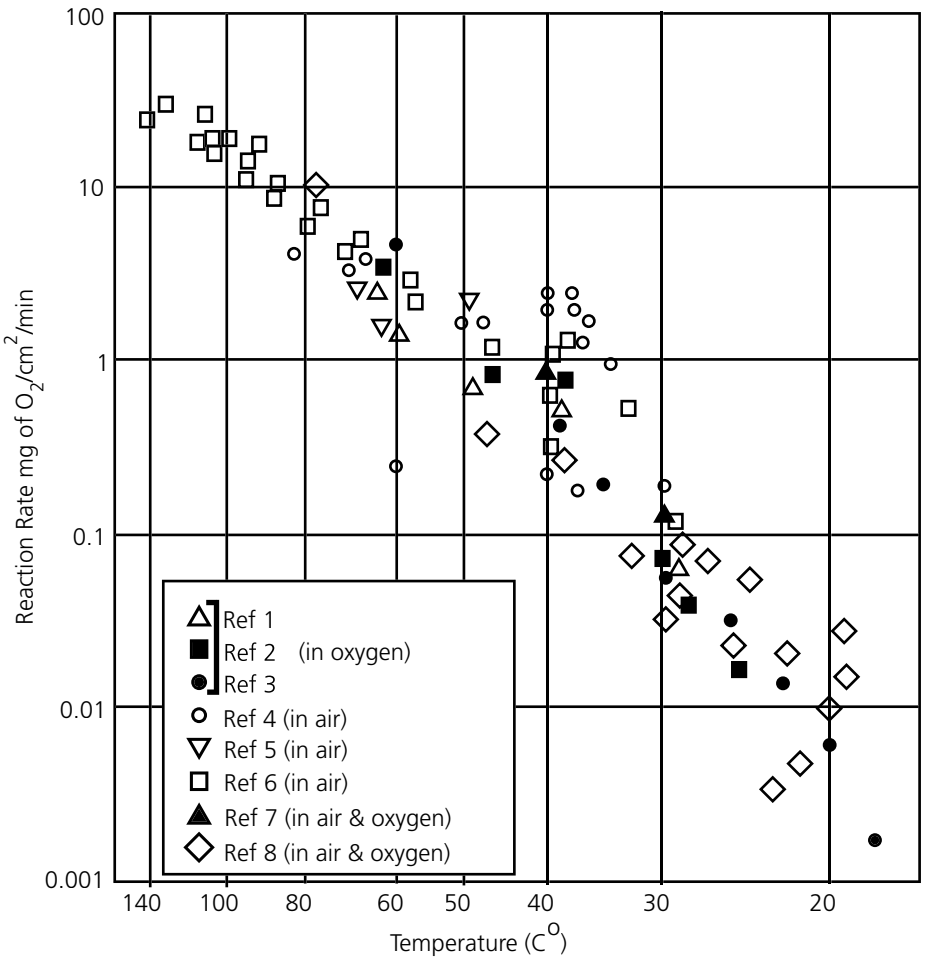
As outlined above, the corrosion and dissolution of metallic uranium or DU can be considered as a two-stage process:
(a) oxidation of zero-valent metallic uranium to U(IV), followed by
(b) oxidation of U(IV) to U(VI).

As is the case for the majority of metals in the periodic table, from a thermodynamic perspective, the first stage in this process is favourable under most Earth surface conditions (ie those in which water and oxygen are usually present). Reaction rates in air and oxygen vary widely from in excess of seven mg of O_2/cm^2 min at 600 °C to less than 0.7 μg of O_2/cm^2 min at temperatures of less than 100 °C (extrapolation from Figure 2).

Whilst oxidation of metallic uranium is thermodynamically favoured, the chemical composition of the oxidant has a fundamental impact on the relative rate (ie the kinetics) of the oxidation process. For example, rates of uranium oxidation in water vapour are much greater than in pure O_2, although the presence of O_2 in water vapour decreases the reaction rate when compared with O_2-free water vapour (Totemeier, 1995). A similar observation has also been made during aqueous corrosion and it has been generally agreed that the presence of dissolved O_2 in reacting waters reduces the corrosion rate of uranium due to the formation of a protective oxide film. The stability of this film is therefore an important factor in the second stage of the corrosion and dissolution process.

Whilst the first stage of the corrosion process outlined above is similar over a wide range of environmental conditions, the second stage is dependent on the prevailing local chemical environment (eg redox and pH conditions), whether this be in soil, air or water, and may be the overall rate-determining step (eg Erikson et al 1990; Wronkiewicz and Buck 1999; Ragnarsdottir and Charlet 2000). In waters rich in bicarbonate and dissolved oxygen (which are often found in shallow groundwaters), even relatively insoluble compounds containing U(IV) have a strong tendency to become oxidised, forming hydrated uranyl minerals and ions (eg $UO_3.H_2O$ (schoepite), UO_2^{2+}, etc). These minerals and complex cations are then free to react with other dissolved inorganic and organic anions (eg chloride, carbonate, bicarbonate, silica, humic acid, fulvic acid, phosphate, sulphate, etc) to form a wide range of complexes (eg Figure 3). Some of these complexes, such as those with silica, may be relatively insoluble and lead to the precipitation of secondary minerals, which inhibit mobility despite the initial formation of relatively mobile species. Hence, it is the relative solubility and geochemical behaviour of these various complexes that typically control the rate at which oxidised uranium may

Figure 2. Reaction rates for the oxidation of metallic uranium in air and oxygen (References from Metals Handbook 1991).



be removed from the corrosion site. This effect can clearly be seen, for example, in the rapid corrosion of unprotected metallic DU in salt fogs and soils (Annexe G) and in the relatively low migration potential of uranium observed in some experiments relating to nuclear waste disposal (eg Wronkiewicz and Buck 1999).

Being highly dependent upon a combination of factors including oxidative-dissolution of uranium, precipitation and dissolution kinetics, and leachant (water) composition, reaction rates for the oxidation of U(IV) to U(VI) and subsequent formation/dissolution rates of complex species are highly variable. For example, oxidation rates for UO_2, or alternatively dissolution rates of $UO_3.H_2O$, can range from tens of days to hundreds of years (eg Braitwaite et al 1997; Wronkiewicz and Buck 1999). Further information on corrosion rates and controlling factors is provided in Annexe G.

3.2.2 Experimentally determined corrosion rates of metallic DU

Whilst it is useful from a mechanistic viewpoint to consider a two-stage process in the corrosion of metallic DU, it is difficult experimentally to study each stage in isolation. Because of this, the corrosion rates in this section are discussed only in terms of the rate of an overall process.

In some experimental cases, particularly those undertaken under field conditions, it is impossible to estimate the effect of corrosion product build-up on corrosion rates. However, such experiments are often undertaken over longer timescales than those in the laboratory and may therefore inherently take into account both the build-up and subsequent transport of products in the immediate vicinity of the DU. Table 4 contains a summary of corrosion rates from studies briefly reviewed in Annexe G.

From the data presented in Table 4 it can be concluded that, typically, corrosion rates in air < water < salt water = soil. However, experimentally determined corrosion rates in air vary markedly with humidity and salt content. From a practical perspective, these data are consistent with observations made during post-conflict studies and those undertaken in proving grounds. For example, during its mission to Kosovo UNEP reported that penetrators found lying on the surface of the ground were often relatively uncorroded, compared with those sampled from within the soil (UNEP 2001). Similar results were obtained during the UK MOD survey of impacted

Figure 3. Eh-pH diagram showing stability fields for uranium under various Eh (in volts) and pH conditions. Eh is an indicator of oxidation potential, and may be related to the presence of dissolved oxygen. pH is an indicator of acidity. Note the wide stability fields (ie the regions bounded by lines) over environmental conditions (moderate Eh and pH) of the dissolved, highly soluble neutral and negatively charged anionic species UO_2CO_3, $UO_2(CO_3)_2^{2-}$ and $UO_2(CO_3)_3^{4-}$, compared with those of the positively charged, strongly sorbed cation UO_2^{2+} and insoluble $UO_2(s)$. The diagram has been constructed for a U-C-O-H system adapted from Brookins (1988) and may be used as a first approximation to predict the chemical form and mobility of uranium species in soils and groundwaters in which Eh and pH have been determined.

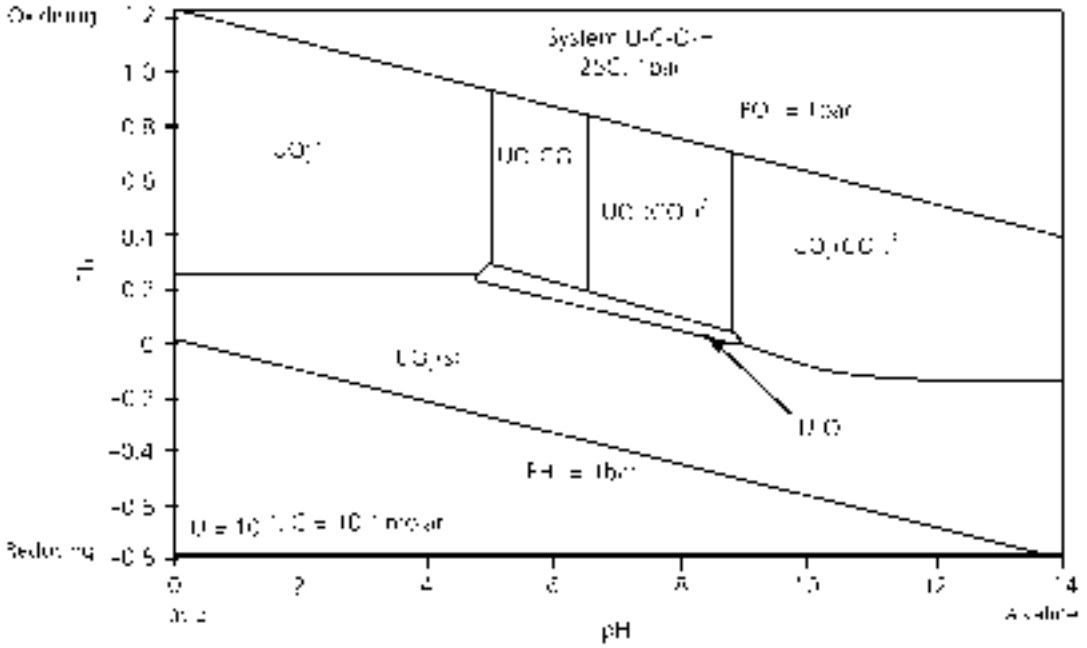


Table 4: Summary of literature corrosion rates (cm/year) for DU-Ti(0.75) alloy

Air	Distilled water	Soil	50 ppm NaCl	3.5% NaCl	5% NaCl
<0.001	0.004	0.050	0.002	0.021	0.077
0.00034		0.100		0.023	
	0.25				
	0.013				

sites (MOD 2001). However, perhaps the most useful evidence (that derived from buried penetrators) is lacking due to the difficulty in finding penetrators once they have travelled further than 30 cm into the soil. Anecdotal information suggests such penetrators have been identified during remediation exercises in Montenegro, but no data regarding their condition have been made generally available at this time. Such information is critical in validating experimental studies of penetrator corrosion discussed in Annexe G, and hence in determining potential impacts of DU corrosion products on groundwater and soil.

3.3 Corrosion and dissolution of dusts

Dusts produced during the impact of DU munitions cover a wide range of chemical and physical forms that depend on the nature of the impact (see Section 2.3.1). Because of this their corrosion rates, or more importantly in this particular context their dissolution rates, are likely to be extremely variable.

Where such dusts are relatively pure U(IV) oxides (which are almost exclusively of low solubility), corrosion/dissolution depends on the rate of oxidation of U(IV) to U(VI). As described above, the rates and products of such reactions are highly dependent upon the local geochemical environment. Because of their high specific surface area such dusts may be considerably more reactive than metallic fragments under similar environmental conditions. Whilst rapid dissolution of such dusts may lead to increased uranium concentrations in soil solutions and pore fluids, it is also likely to promote the precipitation of secondary minerals where this concentration is excessive. In such

cases, it is the solubility of these secondary minerals such as schoepite that controls uranium concentrations in infiltrating water (see also Annexes F and G).

Where dusts produced by impacts are comparatively impure, their corrosion and dissolution behaviour may be markedly different from those of pure U(IV), because of the presence of other elements which may increase, or decrease, the armouring effect of corrosion products.

In general, the corrosion/dissolution rates of such particles are relatively poorly studied compared with the study of the solubility of such substances in biological fluids (an important variable in assessing inhaled dose from radioactive substances). Where corroded or weathered dusts are of a similar physical and chemical form to uranium minerals their solubility characteristics may be extrapolated, although it is rare for naturally occurring minerals to be present as such small particles with such high specific surface areas.

4.0 Environmental pathways

Following the identification of a potential source of contamination, the next step in the investigation of its wider environmental implications is to identify, quantify and model potential environmental pathways by which a specific target such as man may become exposed. This is undertaken not only to assess total levels of potential exposure but also to indicate where controls or monitoring may be most effectively employed to reduce exposure. The objective of this appendix is to review information pertinent to the behaviour of DU through a range of environmental pathways that include transport in air, water and soil to a range of ecosystem compartments and receptors that include agricultural crops, animals and water resources. A key focus is to identify and quantify exposure routes to humans. Various pathways and some possible scenarios by which DU may enter the environment are shown in figure 4.

4.1 Applicability of related studies of uranium in the environment

Although natural uranium and DU differ only in their isotopic composition and would therefore be expected to behave similarly in the environment, they are not derived from chemically and mineralogically similar materials or sources. For example, a military conflict introduces DU directly either onto the surface of the Earth or typically to depths of less than ten meters from the surface (UNEP 2001). It is therefore much more likely that DU will come into direct contact with soils, surface waters and other components of the near-surface environment such as shallow groundwater than, for example, deeper groundwaters that often contain elevated levels of natural uranium.

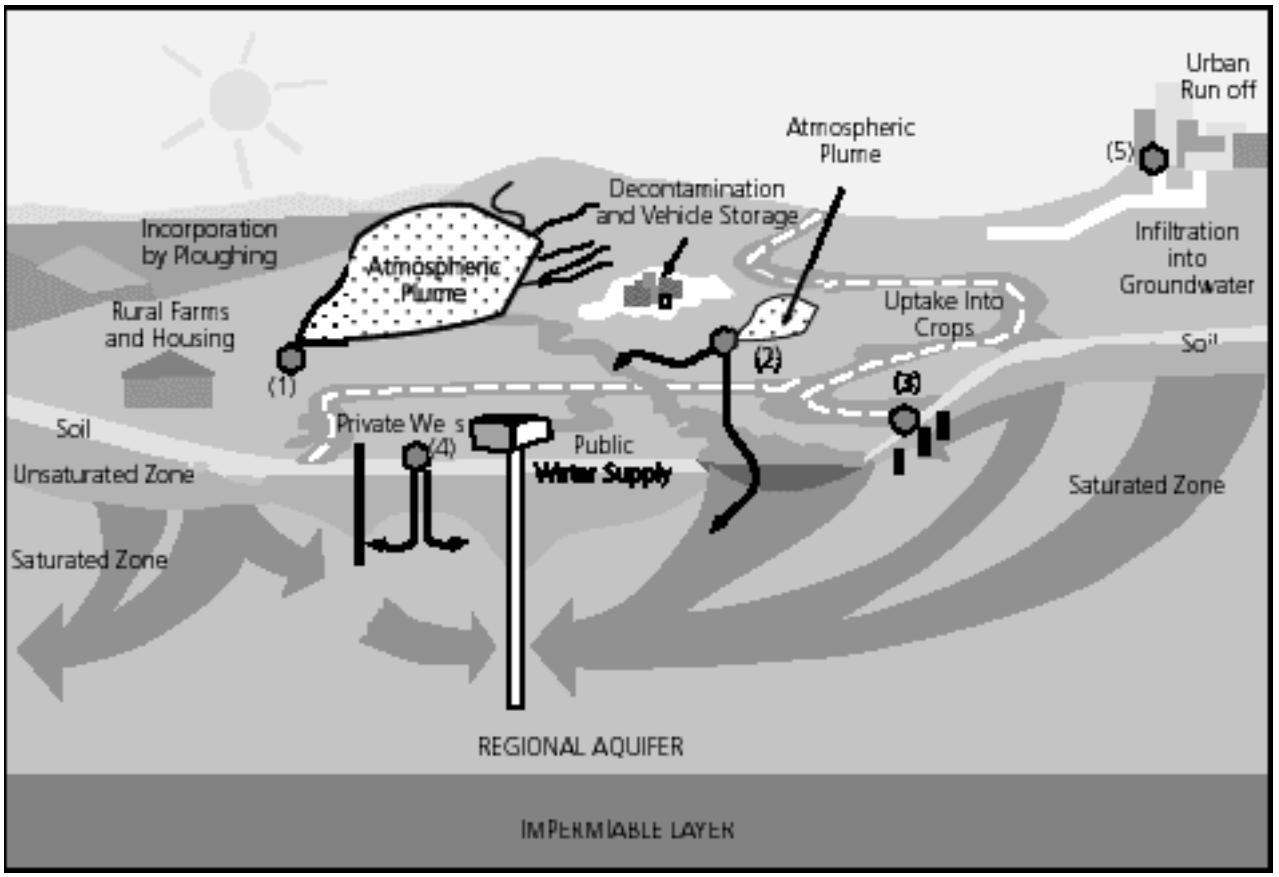
The widest range of available information that might indicate how DU will behave in the environment comes from studies of the behaviour of natural uranium, largely based on studies undertaken for the mining and nuclear industries. However, the applicability of models and scenarios developed for predicting the migration of uranium from nuclear waste - where uranium may be transported into the environment if there is a failure in the surrounding engineered barriers, which often include burial at depth (eg Chapman and McKinley 1987) - is severely limited for military sources of DU. Furthermore, differences in the type of source term complicate the direct extrapolation of data from studies of the behaviour of uranium from mines and nuclear waste disposal to DU in weapons. Military sources of DU are largely present as U-Ti alloys with trace concentrations of fission products (AEPI 1995), whereas in mining uranium is often present as uraninite, and in nuclear waste uranium is present as mixed oxides, within a complex matrix of fission products and other wastes (eg Chapman and McKinley 1987; Wronkiewiez and Buck 2001).

The environmental behaviour of uranium is affected by many environmental variables such as soil composition and chemistry, hydrogeology, resuspension, gut absorption, climate and management. Whilst some authors have suggested that the use of DU munitions are unlikely to add significantly to environmental baseline levels of uranium in soils, it is important to consider that

(1) uranium derived from the fragmentation of munitions may be more bioavailable, and possibly more mobile, than residual natural uranium present in weathered soils (as, for example, demonstrated during investigations of soils contaminated by uranium from the Fernald site by Elless et al (1997) and at military firing ranges by Becker and Vanta (1995)).
(2) the relative importance of additional anthropogenically derived uranium is dependent upon the degree and rate of mixing, and the depth to which such material is incorporated and redistributed amongst the upper soil horizons.

For example, if DU from the impact of a 4.85 kg penetrator (20% volatilised as for the worst-case scenario in Part I of the report) were evenly dispersed over a radius of ten meters to a depth of ten centimetres, it would produce a uranium concentration in soils of approximately 17 mg/kg. This value is above that observed in most natural soils (eg WHO 2001). However, if a similar release of uranium was restricted to the upper one cm or less of soil, as might be expected from the deposition of atmospheric particles onto uniform soils of a high clay content, then the resultant concentration, assuming even airborne dispersal, would be more than a factor of ten higher (ie greater than 170 mg/kg).

Figure 4. Schematic diagram illustrating pathways by which DU may enter the environment Scenarios indicated: (1) tank battle with atmospheric release; (2) air attack on armoured vehicles with limited atmospheric release of DU; (3) air attack in which penetrators directly enter the saturated zone; (4) air attack near water supply wells; (5) use of DU penetrators in the urban environment.



4.2 Air

Background levels of uranium in air vary widely. For example, WHO (1998b) quote values in ambient air from 0.02 ng/m^3 to 0.076 ng/m^3, while in the USA the NCRP quotes a background concentration of 0.30 ng/m^3 (NCRP 1975) and the US EPA a range of 0.15 to 0.40 ng/m^3 in 51 urban and rural areas across the USA (USEPA 1986). During these US surveys it has also been established that ^{234}U/^{238}U ratios vary widely in dust samples (range 0.000054 to 0.00040 as mass abundance or one to seven as an activity ratio, indicating the presence of excess ^{234}U). ATSDR (1999) consider that atmospheric levels of uranium are principally derived from suspension of soils.

4.2.1 Anthropogenic sources

In addition to other carcinogens, tobacco smoke contains significant quantities of uranium and ^{210}Po. Smoking two packs of cigarettes produces in the region of 25 ng of uranium in a form that may subsequently be inhaled (WHO 1998b). Elevated levels of uranium in air (eg three ng/m^3) have also been found downwind of coal-fired power stations associated with their discharges (NCRP 1975). Uranium my also be discharged into the atmosphere from nuclear facilities in which uranium is handled in the preparation and fabrication of fuel assemblies. Data from measurements in the UK indicate annual atmospheric discharges from such sites to be in the range of less than 0.005 to 130 kg (MAFF 1999). Similar releases are documented elsewhere. For example, it has been estimated that airborne releases of uranium at one US Department of Energy facility amounted to 310,000 kg between 1951 and 1988 (equivalent to a rate of approximately 8000 kg per annum). This produced an estimated offsite inventory of 2130-6140 kg of excess uranium in the top five centimetres of soil in the vicinity of the facility (Meyer et al 1996). Other data from the USA and Canada have also shown elevated uranium levels in and around milling and processing facilities, measured values ranging from three to 200 ng/m^3 at distances of up to two kilometres from site boundaries (ATSDR 1990;1999).

Data relating to the concentration and transformation of uranium and its compounds in air and their bioavailability were cited as being required for future studies in a toxicological assessment of uranium by the US Department of Health and Human Services (ATSDR 1990). While this has been supplemented in the USA by additional collection of data (ATSDR 1999), data from other countries remain limited.

Table5. Summary of air concentrations and fraction aerosolised from DU penetrator impacts

Report	Mass concentration (mg/m^3)	Fraction of penetrator aerosolised (%)
Reports obtained		
Hanson et al (1974)	500–1700 (exit chamber) 70–600 (entrance chamber)	0.25[1]
Glissmeyer and Mishima (1979)	8–35	70
Chambers et al (1982)	130 (average)	3 (1.5–5)
Brown (Personal communication 2000)	13–60 (inside, at 3 m) 7–17 (outside, at 7 m)	
Reports not obtained (data from OSAGWI 2000, tab L)		
Gilchrist et al (1979)	Near target, >0.3 for 5 min and >15 min (dry surface); but <15 min (wet surface)	17–28
Fliszar et al (1989)[2]	44,400 (initial, inside tank)	8.5
Jette et al (1990)		<10 (0.02 – 0.5)
Parkhurst et al (1990)		<10

[1]*Not assessed by authors. Calculated from concentration and volume of enclosures (see Annexe G, Royal Society 2001)*
[2]*Report subsequently obtained.*

4.2.2 Airborne DU following armed conflict

Part I of the report reviewed the concentrations of aerosolised DU from a number of references. These are summarised in Table 5.

Measured mass concentrations close to the target are very high initially: up to 1700 mg/m^3 in tests using pieces of armour plate, but up to 45,000 mg/m^3 in the test using a tank (Fliszar et al 1989). However, the concentration drops rapidly. In the study by Glissmeyer and Mishima (1979) it fell from 8 to 35 mg/m^3 to less than one mg/m^3 within ten minutes. These trials were, however, conducted in the open, and so the aerosol could easily disperse.

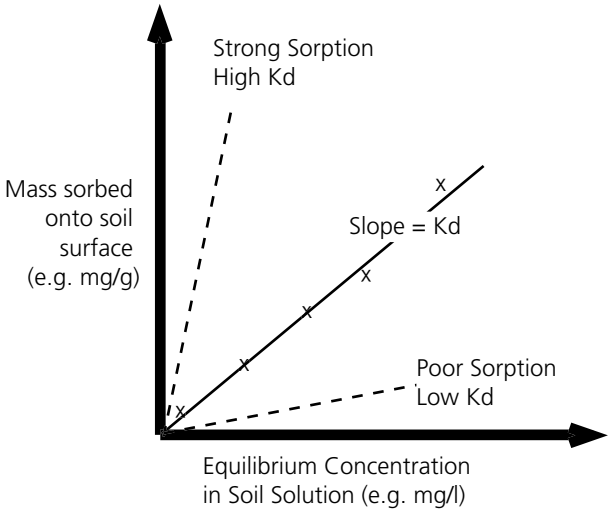
Estimates of the fraction of the penetrator aerosolised can also be used to estimate the initial concentration, on the assumption that the aerosol is dispersed uniformly inside the vehicle. Here the vehicle is assumed to be a box of dimensions three by two by two meters, having a volume of 12 m^3. In a worst case, assume that 20% of a five kilogram penetrator is dispersed: ie 1000 g in a volume of 12 m^3, giving an initial concentration of about 100,000 mg/m^3. Consider as a more typical central estimate that 100 g is dispersed (ie 2% of a single five kg penetrator, or 10% of three 0.3 kg penetrators). This would give an initial concentration of about 10,000 mg/m^3.
CHPPM (2000) (page 151) reports unpublished test data showing that the concentration inside a tank fell by about a factor of ten every ten minutes (falling to about 0.02% of the initial amount at 30 minutes). Therefore it is assumed here that the initial concentration is maintained for one minute, that it is a factor of ten lower for ten minutes and a further factor of ten lower for ten minutes, and so on.

Bou-Rabee (1995) measured uranium concentrations and isotopic ratios in eight air samples collected following the Gulf War (sampled in 1993–1994). The observed concentrations varied between 0.22 and 0.42 ng/m^3 with $^{235}U/^{238}U$ ratios ranging between 0.005 and 0.007. A broadly similar exercise was also performed after the Kosovo conflict to investigate if DU could be detected in airborne particles from Hungary (Kerekes et al 2001). Whilst no characteristic signature of DU could be detected by alpha spectrometry, elevated levels of uranium with a natural isotopic signature were observed during the conflict and these were attributed to well-dispersed dusts (2.5 microns in size) emitted into the atmosphere during bombing (supported by the geographical and temporal distribution of measured concentrations). This study emphasises the potential for long range transport should a large proportion of DU be converted to dust as a result of high energy hard target impacts occurring during a military conflict. The results are also consistent with observations from Kosovo (eg UNEP 2001, MOD 2001 and other personal communications), suggesting that production of such dusts during the conflict were minimal.

4.3 Soil

The absolute concentration of uranium and its mineralogical associations in soil vary widely, reflecting the abundance of uranium in the parent geological materials from which the soils were formed, soil

Figure 5. Example of a linear isotherm illustrating the derivation of the K_d term. The x-y plot is of the concentration of contaminants such as uranium sorbed onto the soil versus the equilibrium concentration of the contaminant in associated soil water, where the slope of the resultant line is equal to K_d.



development processes in which uranium may become concentrated (ie in organic-rich horizons) or leached, and the addition of uranium from anthropogenic activities.

The mineralogical form of uranium found in soils depends upon that present in parent materials (rocks and associated mineral-bearing horizons) and the evolutionary history of soil formation. Soils developed over granitic rocks may contain a significant proportion of their associated uranium content trapped within resistant soil minerals such as zircons for millions of years, whilst other soils in which uranium is actively being absorbed from up-welling groundwaters may contain significant proportions of relatively soluble secondary uranium minerals. Even where mobile uranium may exist with soil fluids, significant sorption onto clay and organic matter can significantly affect mobility within specific soil horizons (eg Harmsen and de Haan 1980; Read et al 1993).

Uranium (VI) phosphates and silicates such as autunite, soddyte and uranophane have been found in uranium-contaminated soils (Buck et al 1996; Morris et al 1996). Studies of dispersal of uranium at natural analogue sites have demonstrated that oxides of U(IV), including uraninite and pitchblende, may be readily weathered by oxidation and complexation with inorganic and organic ligands and converted to more mobile, soluble forms of uranium (Burns and Finch 1999).

The mobility of uranium in soil affects the extent of plant uptake and groundwater contamination. It is strongly controlled by the proximity of groundwater to the soil environment, soil and water pH, soil organic carbon content and, to a lesser extent, the abundance of cation exchange sites such as those found on clays (eg Ribera et al 1996; Burns and Finch 1999; USEPA 1999, 2000). Unlike many heavy metals, such as lead, the mobility of uranium is higher in moderately alkaline soils compared with acidic soils, due to the formation of stable negative complexes (oxy-anions) with oxygen and carbon. Thus, uranium sorption values are low in moderately alkaline soils, rich in montmorillinite (a clay mineral with a high cation exchange capacity) but low in organic carbon, such as those soils occurring in Western Turkey and other semi-arid Mediterranean-type environments (eg Zielinski et al 1997; Akcay 1998). Similar observations on the effect of pH on uranium sorption and mobility were observed by Erikson et al (1990), during studies of soils from the Aberdeen and Yuma Proving Grounds in the USA (K_d of 54 ml/g at pH 8.0).

The mobility of a dissolved component within soil pore water is controlled by sorption. This is a general term covering processes occurring at the solid-solution interface including specific adsorption (eg cation exchange) and non-specific adsorption. The most commonly used indicator of sorption or pollutant mobility is the soil water distribution coefficient (K_d, commonly defined as the concentration of a given substance in solution divided by the concentration sorbed to soil constituents). The K_d represents a special case 'linear' isotherm (see Figure 5) (Domenico and Schwartz 1990).

The organic carbon content of a soil strongly influences both pH and the soil's ability to sorb uranium. Hence, soils with high organic carbon content generally have a high K_d for uranium. For example, enrichment factors (concentration in peat divided by concentration in soil) of between 200 and 350 have been cited for absorption of uranium onto peat (Horrath 1960). Values of K_d for uranium for various soil pH values are given in Table 6.

Table 6. Ranges of K_d for various soils based on pH (USEPA 1999); higher values indicate greater sorption and hence lower mobility (see also Annexe F)

Soil pH	3	4	5	6	7	8	9	10
K_d ml/g (minimum)	<1	0.4	25	100	63	0.4	<1	<1
K_d ml/g (maximum)	32	5,000	$1.6x10^5$	$1x10^6$	$6.3x10^5$	$2.5x10^5$	7,900	5

Note: Typical Northern and Central European soils have a pH range of 5 to 7 whilst those in Mediterranean environments and formed over limestones typically exhibit pH ranges of 7 to 9. Additions of various soil conditioners and fertilisers such peat, lime or phosphate may significantly affect the behaviour of uranium in soils.

Despite extensive tabulations of K_d data (eg Annexe F) and their utility for describing sorption in mathematical models, K_d is inherently a site-specific value which is only valid across a specific range of pollutant concentrations for which the assumption of a linear relationship (isotherm) holds and a specific range of pollutant chemistries. Differences in pollutant and soil chemistry result in wide variations in reported K_d values even when pH is taken into account (eg Table 6). The use of geochemical modelling codes such as PHREEQC (Parkhurst and Appello 1999), or coupled chemical transport codes, in which predictions concerning the physical migration of uranium are coupled to chemical processes that may retard such migration, offer a better predictive capability. However, to realise their advantages such models inherently require extensive site-specific data and expertise, which are often unavailable.

Furthermore, in using K_d values a careful judgement must be made as to whether it is perhaps more appropriate to use a conservative approach and to allocate a K_d value of zero to a given situation. This approach is often recommended where migration is likely to impact directly on a sensitive receptor (eg a frequently used water well) or where sorption sites may become saturated (eg Gillespie et al 2000). There should be no differences between the values of K_d for DU and uranium because of their chemical similarity, although the value of the K_d does change with the chemical form of uranium (or DU) present.

Despite uncertainties associated with the use of K_d values on a site-specific basis, an understanding of processes associated with sorption of uranium allows the identification of regions or areas in which the mobility of uranium or DU is likely to enhanced. For example, using data presented in Table 6 and a worldwide map of soil pH it is possible to indicate areas of potentially enhanced mobility (Figure 6). Similarly, maps of soil organic carbon content can be used to highlight areas of low or enhanced mobility.

Although the corrosion and weathering rates of DU oxides and metallic DU are low (Section 3), they are still relatively rapid processes compared with those of uranium in many natural soil minerals. As for natural uranium, the mobility of weathered DU in the soil profile is dependent upon sorption and mass transport properties of the soil (ie K_d and the infiltration rate of water). The variation in K_d for uranium with organic carbon content and soil pH indicates that mobility is likely to be greater in semi-arid calcareous environments, or calcareous environments in which neutral to alkaline soil pH combines with a low organic carbon content. Uranium has been shown to be mobile in environments subject to high surface erosion and low infiltration rates, such as deserts, for example in Israel (Gross and Ilani 1987; Gill and Shiloni 1995), Jordan (Smith et al 1996) and the USA (Zielinski et al 1997). Whilst mobility is greater in semi-arid, calcareous soils, low net infiltration due to the lack of precipitation and high evapotranspiration may significantly reduce the transport of DU.

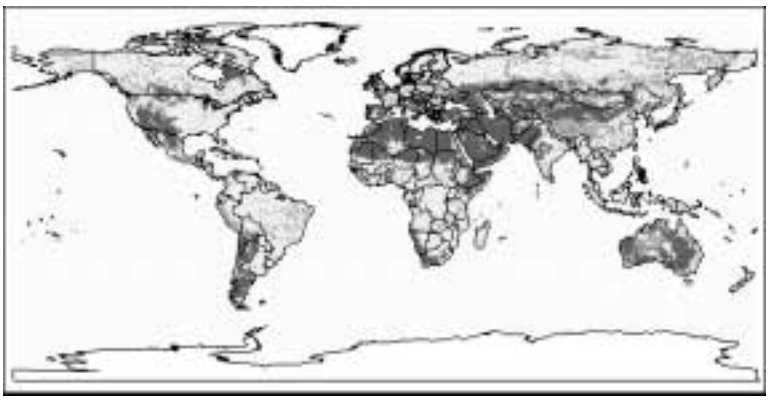
The enhanced mobility of DU in a given soil type potentially leads to both positive and negative outcomes that need to be evaluated on a case-by-case basis. Enhanced mobility has a potentially negative impact on groundwater and a similarly detrimental effect on the cost and technical feasibility of clean-up. However, it may also be advantageous in dispersing point source pollution events (thereby reducing exposure of soil compartments, including biota, to 'hot spots') and significantly reducing the concentration of DU in resuspended material.

4.4 Surface and groundwater

4.4.1 Surface water

Uranium is present to some extent in all natural surface waters as a result of the weathering of soils and rocks that contain natural uranium. Studies of the abundance of uranium in over 120,000 UK surface waters indicate a log-normal distribution with a mean of 0.65 ppb and a 95th percentile of two ppb. This range of values is consistent with concentration data collected from elsewhere in the world (Ivanovich and Harmon 1982; ATSDR 1999; WHO 2001). Sea water contains approximately three ppb of dissolved uranium, derived from the weathering of terrestrial rocks, the exact concentration varying linearly with salinity (eg Ivanovich and Harmon 1982).

Figure 6. Worldwide distribution of soil pH (adapted from a database of soil pH at a scale of 0.5 by 0.5 degrees (Batjes 1996)) thematically shaded to indicate surface soils (0 to 30cm) in which uranium is likely to be highly mobile (dark grey), moderately mobile (medium grey) and of restricted mobility (light grey to white).



In surface water uranium may be present as, or sorbed to, particulate or colloidal material, particularly where such materials contain naturally occurring organic materials such as humic and fulvic acids (eg Choppin 1992; Higgo et al 1993; Ragnarsdottir and Charlet 2000). When in solution, stream water acidity and alkalinity, Eh and organic carbon content play an important role in controlling aqueous phase speciation and mobility of uranium (Annexe F).

Exposure of surface water to DU contamination is likely to be dominated by transfer from direct soil deposition in catchments (Ebinger et al 1996), where firing occurs over land, but may also occur directly or through the introduction of DU from contaminated groundwater (via springs and baseflow). The loss of DU from catchments to water bodies will be controlled by physical and chemical processes as described above, or through the physical transport of DU in runoff. For example, overland water flow, from rainfall or snow thaw, will cause the physical movement of particles to surface watercourses, and ultimately into estuaries and coastal areas. The migration of uranium-rich particles by this process is well established and forms the basis of geochemical exploration and mapping.

4.4.2 Groundwater

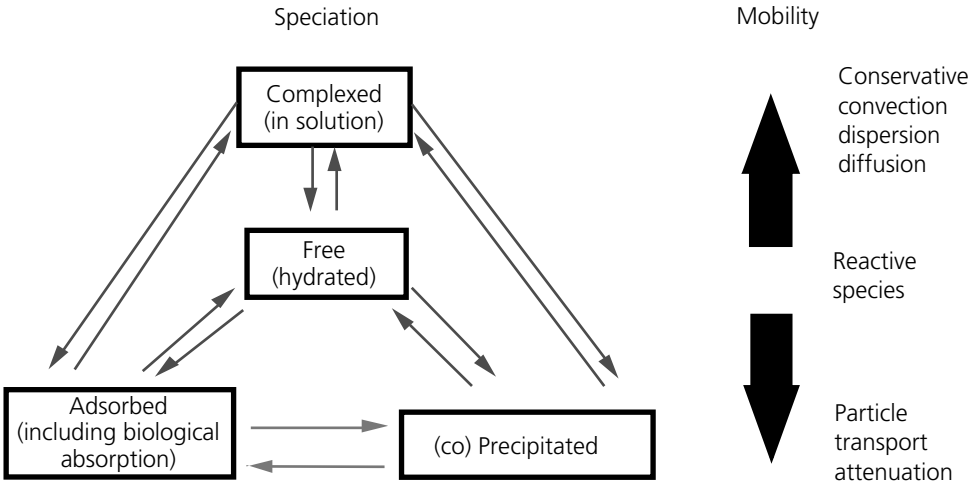
Uranium is present to some extent in all groundwaters as a result of the weathering of rocks, which themselves contain natural uranium. Concentrations of uranium are generally higher in groundwater than in surface water and are highly variable, depending on the presence of uranium in associated parent materials (ie rocks), the ease by which it may be released from its geological source or proximity to industries that may introduce uranium into the environment. Waters, particularly those whose major element chemistries promote uranium solubility and mobility (eg typically oxic (high positive Eh), neutral to moderately alkaline with a high bicarbonate content), have higher uranium concentrations, and concentrations of greater than 1000 ppb have been observed in a number of such aquifers (WHO 1998b, 2001). It is not unusual to encounter groundwaters containing between one and five ppb uranium in aquifers whose host rocks contain relatively low concentrations of uranium (eg those derived from aquifers developed in limestone often contain higher dissolved uranium concentrations than those derived from areas of granitic rocks).

When considering the vulnerability of groundwater to pollutants sourced on the Earth's surface, the soil zone is considered to act as a protective layer in which pollutants are filtered from infiltrating water. This is principally because sorption in the unsaturated zone and aquifer is much more limited than in soils where organic carbon and clays may act as extremely efficient sorbants. Discussions relating to sorption of uranium in soils in Section 4.3 are therefore important considerations when evaluating groundwater vulnerability.

The single most important, often overlooked, factor controlling the vulnerability of groundwater, assuming that uranium is mobile, is the depth of the unsaturated zone (ie the proximity of the contamination to the water table) and the infiltration rate. The vulnerability of water resources derived from river gravels may be high due to their proximity to the surface, whilst that of water resources obtained from deeper, possibly confined, aquifers will be inherently lower. Because of this, knowledge of the potential penetration depth of munitions into the surface environment is a very important factor in assessing any potential impact of the use of such weapons on groundwater or indeed surface water reserves, as the penetration increases the proximity of groundwater to the penetrator and potentially bypasses the soil zone in which sorption is most likely to take place.

As described above in Section 4.3, the mobility of dissolved DU in soil is controlled by factors such as the pH of soil minerals and water, and the sorption capacity of soil minerals. Thus, where soil strongly binds DU to minerals or on surfaces (eg iron oxides, clay minerals or

Figure 7. The effect of chemical speciation on mobility and sorption processes (adapted from Bourg (1988)). In the case of uranium, complexed species are often particularly mobile due to the formation of oxy-anion complexes with zero or negative charges.



organic carbon), its release into soil water, and translocation to groundwater, should be minimal. In deeper environments, mobility and attenuation are controlled by the composition of fracture coatings and water chemistry. Where uranium is highly mobile, water resources may be more vulnerable to contamination.

In addition to the transport of DU in the aqueous phase, physical translocation of particulate material into groundwaters may occur, through the regolith[1] and within aquifers, which exhibit both primary and secondary fracture flow.

If DU is poorly sorbed in soils and reaches the unsaturated and saturated zones, its concentration will depend more upon the physical rather than the chemical properties of the aquifer. For example, in a rapidly flowing aquifer DU in infiltrating surface waters will undergo potentially significant dilution. Similarly, dilution effects are also more likely to occur in an aquifer with high dispersivity rather than in a fracture flow network that has an inherently low dispersivity.

4.5 Micro-organisms

Micro-organisms play an important role in many fundamental environmental cycles, such as the recycling of organic matter in soils, and often represent important fundamental sources of biomass and nutrients at the base of food chains. Because of this, protection of microbial diversity and function underpin many environmental assessments. Their position at the base of many food chains has also encouraged their use as sentinel organisms with which to predict the potential for environmental harm. Micro-organisms have also evolved mechanisms to inhibit or promote the absorption of potentially toxic trace elements and consequently they have been studied within the context of bio-remediation and bio-leaching of metalliferous ores, and the migration and attenuation of pollutants associated with radioactive waste disposal.

4.5.1 Toxicity

Harm to micro-organisms exposed to DU may result either from its chemical properties or its inherent radioactivity. The radioactivity associated with uranium (and even more so in the case of DU) is considered to be non-lethal to micro-organisms due to the long half-life of uranium and the short life cycle of micro-organisms (Ehrlich 1996). The chemical properties of uranium may, however, induce significant toxic effects, similar to those caused by other metal ions (including heavy metals).

The degree of toxicity of a metal ion to a micro-organism (typically measured by the damage the metal ion can do to the cell) varies in a similar way to toxicity to higher forms of life (ie with the type of ion, its chemical speciation and concentration). Many elements such as copper and nickel may be both essential at low concentrations and toxic at elevated concentrations. Uranium or DU have no known biological function in micro-organisms and are considered to be potentially harmful to single cellular species even in low concentrations. For example, studies of the degree of resistance of ten different isolates of *Thiobacillus ferrooxidans* to the metals Cu, Ni, U and Th showed that uranium is 20 to 40 times more toxic than either copper or nickel at pH 2.1 (Leduc et al 1997). Similarly, uranium exhibited a stronger inhibitory effect on the growth of *Pseudomonas aeruginosa* and *Citrobacter* spp. than Th (Premuzic et al 1985; Plummer and Macaskie 1990). The uranyl ion has been shown to affect *Thiobacillus ferrooxidans* by inhibiting iron oxidation and carbon dioxide fixation (Tuovinen and Kelly 1974a,b). However, toxicity may occur in different ways in different organisms.

[1]Regolith is the irregular blanket of loose particles that cover the Earth and include soil, alluvium, and rock fragments weathered from the bedrock.

The degree to which uranium influences cellular function also depends upon the ability of the micro-organism to control the local concentration of specific ions. For example, cells may differ in their ability to prevent passage of toxic ions through the cytoplasmic membrane, to pump ions out of the cytoplasm, and to sequester ions from solution by adsorption and precipitation. Factors affecting the toxicity and bioavailability of uranium are similar to the factors (pH, chemical speciation) affecting uranium biosorption (Tuovinen and Kelly 1974a,b; DiSpirito and Tuovinen 1982; DiSprito et al 1983). In addition to these factors, solution redox state, sorption onto inorganic and organic compounds, and complexation by organic compounds are also considered to be important in the natural environment.

As discussed earlier in Section 4, ferric oxyhydroxides, organic materials and natural organic ligands may be very important potential sorbents of uranium. Both ferric oxyhydroxides and organics commonly occur in sediments and soils with high capacity for uranium sorption (Tripathi 1983; Hsi and Langmuir 1985; Wood 1996; Langmuir 1997) and can reduce the bioavailability of U(VI) to micro-organisms (Gadd and Griffiths 1978; Babich and Stotzky 1980; Gadd 1993). Similarly, complexation with organic molecules present in the environment has been shown reduce the bioavailability of uranium in *Citrobacter* spp. (Young and Macaskie 1995). In dry biomass (*Myxococcus xanthus*) absorption was considered to be rapid, strongly influenced by pH and reversible on the addition of sodium carbonate. The sites of absorption were identified as the cell wall and within the extracellular polysaccharides of this micro-organism (Gonzalez Munoz et al 1997).

Meyer et al (1998b) used a soil microcosm to investigate the impact of uranium on a range of soil functions. Soil respiration, which represents the overall soil biological activity, was found to be the most sensitive measure of functional changes. At concentrations above 500 mg/kg there was found to be a significant decrease in soil respiration. At concentrations of 25,000 mg/kg the decomposition of organic litter was also affected. In particular it was noted that the decomposition of lower quality litter was much more greatly affected than that of high-quality litter, which is consistent with the behaviour of other heavy metals.

4.5.2 Accumulation

Certain microbial species accumulate uranium. Biomass from filamentous fungi such as *Aspergillus niger*, *Rhizopus oryzae* and *Penicillium* spp., yeasts such as *Saccharomyces cerevisae*, algae such as *Chlorella regularis*, actinomycetes such as *Streptomyces longwoodensis* and unicellular bacteria such as *Citrobacter* spp., and *Pseudomonas aeruginosa* are capable of uptake or binding of uranium to greater than 15% of dry weight biomass (Hu et al 1996). Uranium biosorption mechanisms vary and include complexation, ion exchange, co-ordination, adsorption, chelation and microprecipitation. The ability to bind uranium has prompted extensive studies to investigate the potential of such microbes as agents to remove uranium from liquid wastes.

The ability to grow in the presence of elevated metal concentrations is found in a wide range of microbial groups and species, and micro-organisms from sites highly contaminated with uranium have been shown to accumulate far greater quantities of uranium than those from uncontaminated sites. Suzuki and Banfield (1999) have suggested that this is due to an adaptive increase in tolerance. Whilst such increases in tolerance and incorporation of uranium without adverse effect suggests a lower specific toxicity amongst some microbial species, it also represents a route by which uranium may become concentrated within micro-organisms that may form a significant niche within a number of food chains.

4.6 Plants

Plants are generally poor accumulators of uranium and concentrations of uranium in plants are generally several orders of magnitude lower than those in the soil in which they grow. The uptake of uranium by plants, although low compared with mobile radionuclides such as radiocaesium and radiostrontium, is higher than for other actinides. For instance, Garten (1980) reported higher uptake of ^{234}U and ^{238}U into leaves of boxelder trees than of Cm, Am or Pu. Despite the generally low transfer of uranium from soil to plants, certain plant species exhibit a high uptake of uranium.

4.6.1 Species differences and biodistribution

In general, uranium concentrations in non-vascular plants (mosses and lichens) are higher than those in vascular plants (Cramp et al 1990). These plants have been used as indicators of uranium contamination, for instance, around uranium mines, such as in the study by Beckett et al (1982) that recorded decreasing uranium concentrations in moss and lichen with increasing distance from a mine. Similarly, UNEP have used lichen and bark as bioindicators of atmospheric DU contamination (Sansone et al 2001; UNEP 2001).

High accumulators of uranium have been reported within different plant groups. High transfer of uranium has been reported in old black spruce twigs and some boreal forest plants, in addition to lichens and moss (Thomas 2000a). The high $U/^{226}Ra$ ratios in old black spruce twigs compared with all other vegetation was thought to be due to the deep root system of this species, which would enable enhanced uptake of uranium from groundwater. Within crop species, Evans and Erikkson (1983) identified sugar beet tops as high accumulators of uranium.

4.6.1.1 Distribution within plants
In sphagnum mosses, uranium concentrations have been reported to be significantly higher in the lower, brown parts of the moss compared with the upper green part (Sheppard et al 1984). In vascular plants, roots generally have higher uranium concentrations than aerial parts of the plants. There is considerable accumulation of uranium in plant roots of some species. Translocation of uranium from roots to other parts of plants seems to be small. In general, uranium concentrations in plants decline in the order:

roots> shoots> fruits and seeds

Early observations (Acque (1912) and (1913), quoted in Sheppard (1980)) noted that uranium formed yellow deposits in the cell nuclei of root meristems, which then prevented translocation. Vinogradov (1959) stated that insoluble calcium uranyl phosphates are deposited on root surfaces, allowing only a small amount of root uptake. Uranium has been shown to deposit as electron-dense crystals on oat and barley roots exposed to uranyl acetate (Wheeler and Hanchley 1971; Robards and Robb 1972). Initial deposition of uranium occurs in the cell walls in the root cap and meristematic zone, and subsequent migration into the plant protoplast may occur by pinocytosis. Plant products secreted by roots also bind uranyl ions onto root surfaces and may inhibit uranium uptake by roots.

There are exceptions to the general trend of higher uranium concentrations in plant roots (for a detailed discussion see the review in Cramp et al (1990)). In most plant species, including arable crops, uranium concentrations in seeds are lower than those in stem, stalk or straw. For crops, there are fewer reported exceptions to the trend above than for uncultivated plants, but they do occur. For instance, Prister (1969) reported 2.7-fold higher uranium concentrations in carrot leaves than in roots in plants grown in a well-cultivated soil.

Various relationships have been reported between uranium concentrations in plants and those of other elements, although the mechanism determining these relationships is not clear. However, some relationships may be explained by the uranyl ion seeking oxygen-binding molecules, as does Ca^{2+} and Mg^{2+}. UO^{2+} forms more stable complexes with phosphates and carboxylic acids and other oxygen-containing ligands than Ca^{2+} and Mg^{2+}. The uranium distribution in a wide range of plant types was reported by Prister (1969) to be inversely related to the ash content minerals. Plant species with high concentrations of Ca and K have been shown to contain low uranium concentrations (Prister 1969; Evans and Eriksson 1983). A strong positive correlation was reported between uranium and P concentrations in leafy vegetables by Morishima et al (1977), but the relationship was much weaker for uranium and Ca. Plant species with a cell sap pH of less than 5.2 were shown to readily absorb uranium (and Ca, S, Se, Na but not K) by Cannon and Kleinhampl (1956).

4.6.1.2 Bioremediation
Very high accumulation of uranium in roots has been reported in certain plant species such as sunflowers (eg Dushenkov et al 1997). This has led to the suggestion that these high accumulating species can be used for bioremediation of highly contaminated soils.

4.6.1.3 Time dependency
Uranium concentrations in plant tissues may change with time. For instance, Dunn (1981) reported that uranium accumulation occurred in spruce twigs when they were actively growing, to a maximum at two to four years old and then declining with age.

4.6.2 Exposure pathways
Contamination of plants by uranium can occur via a number of different routes, the two most important of which are: from the atmosphere (ie wet and dry deposition to foliage) and via uptake through membranes (soil solution, irrigation water or rainfall), through resuspension of soil-associated uranium.

4.6.2.1Wet and dry deposition of atmospheric particulate material
Uranium present in the atmosphere can be deposited on plant and soil surfaces. No information has been found on foliar uptake of uranium. Uranium present in the atmosphere will normally be due to resuspension of soil. Atmospheric deposition on plant surfaces and soil is the most likely mode of contamination when metallic DU is converted into dusts or aerosols, for instance after the impact of weapons with armoured targets or following an intense fire in which DU is present. Surface contamination by uranium may be minimised through thorough washing of vegetables, greens and fruit.

4.6.2.2 Root uptake
The extent of root uptake of uranium is principally controlled by the mobility of uranium in the soil solution. The extent to which uranium or DU is sorbed to soil components, and the strength of that binding, affects the amount of uranium that is in soil solution and thus in plants. If the binding of uranium to soil components is weak, depletion of uranium in the soil solution will lead to dissociation of bound uranium and replenishment of the solution.

Plants can absorb soluble forms of uranium, however; in many soils uranium is strongly sorbed and can be present in a highly heterogeneous pattern in soils with poor root contact. Since uranium is quite immobile in many soils, any mechanism that increases mobility is important in enhancing root uptake, including the formation of complexes and associations with colloids (see Section 4.2).

Table 7. Concentration ratios for uranium for different plant groups (WHO 2001)

Plant group	Concentration ratio (Bq/g per Bq/kg dry soil)		
	Weight basis	Minimum	Maximum
Leafy vegetables	Fresh weight	1.2×10^{-4}	1.0×10^{-2}
Root vegetables	Fresh weight	2.0×10^{-4}	3.0×10^{-2}
Fruits	Fresh weight	4.0×10^{-4}	4
Grains/cereals	Dried weight	2.0×10^{-4}	1.3×10^{-3}
Pasture grass/browse	Dried weight	1.0×10^{-5}	0.2

The UK's National Radiological Protection Board (NRPB) uses a CR for all vegetables and pasture grass of 1×10^{-3} (NRPB-R273), based on fresh plant weight to dry weight soil, within their FARMLAND model and GDL assessments.

Few studies have compared the mobility of different uranium isotopes, but Evans and Eriksson (1983) showed that transfer between plants and soil for ^{234}U and ^{238}U was similar for a wide range of different crops in Sweden. Uptake of both uranium isotopes was much higher in sugar beet tops than in the other crops.

The depth at which uranium occurs in soil has been shown to affect plant uptake in sandy soils, but not in loam soil in experimental studies where uranium was placed at different depths in the soil profile (Sheppard et al 1984). In sandy soil, much more uranium was taken up by alfalfa and chard from uranium placed near to the soil surface, implying that uptake was dependent on root activity, although there may have been reduction of uranium to less mobile forms with depth. This might suggest that DU deposited on soil surfaces may be more bioavailable than uranium dispersed throughout a soil profile. However, the physical and chemical form of the DU is likely, at least in the early period after deposition, to differ from those of natural uranium and this may mask such effects.

4.6.3 Quantification of transfer from soil to plants

Transfer of metals or radionuclides from soil to plants is commonly quantified using the concentration ratio (CR), defined as the concentration in the dry plant (eg mg/g or Bq/kg) divided by the concentration in the dry soil (eg mg/g or Bq/kg). For many radionuclides, it is assumed that the CR is a constant for a specified radionuclide source, type of soil and plant species, and that the plant and soil concentrations are linearly related with the line defining the relationship passing through the origin. For uranium, this represents a conservative assumption for cases where soil uranium levels are elevated (see discussion below on data of Sheppard and Evenden (1988a,b)).

CR values for different types of plant have been compiled and examples of CR for uranium are given in Table 7. In their review of CR values, Sheppard and Evenden (1988a,b) reported that comparison of plant types showed significantly higher values for root crops than for fruit, cereals, shrubs or leafy vegetables. According to a recent review by Thomas (2000a,b), plant-soil CR values for uranium are generally in the range 10^{-5} to 10^{-1}, depending on species, tissue and soil.

The UK's National Radiological Protection Board (NRPB) uses a CR for all vegetables and pasture grass of 1×10^{-3} (NRPB-R273), based on fresh plant weight to dry weight soil, within their FARMLAND model and GDL assessments.

In a review of CR values, Sheppard and Evenden (1988a,b) reported that the CR for uranium decreased as the corresponding soil uranium concentration increased. To overcome this problem, they statistically adjusted the CR values derived for different food crops to correspond to a soil concentration of five mg uranium per kg. This allowed a direct comparison of the differences between species and is shown in Table 8. The CR for other uranium concentrations in soil could then be calculated using the equation

$$\log_{10}(CR_{unknown}) = \log_{10}(CR_{table}) - 0.629 \times (I_{soilU} - 0.690) \quad (1)$$

where I_{soilU} is the $\log_{10}$ of the required soil uranium concentration in g/g.

In their review of CR values, Sheppard and Evenden (1988a,b) reported similar values for three soil types, but fine soils gave significantly lower CR values than did coarse, peat or tailings soils. In a separate review, Cramp et al (1990) concluded that plant uptake of uranium from sandy soils is greater than that from clay or loam soils. In a study by Thomas (2000a), the behaviour of uranium in a bog and a pine habitat were compared. In contrast to other observations, he found lower uptake from sandy topsoil compared with peat and lower extractability in sand.

4.6.4 Is uranium an essential trace element in plant metabolism?

Early literature reported that uranium was probably an essential element for higher plants (summarised in Dinse and LaFrance (1953)). This was based on observations of a

Table 8. Weighted average CR values for uranium adjusted to a mean soil concentration of five mg uranium per kg (after Sheppard and Evenden (1988a,b))

Plant type	Soil type				
	Fine	Coarse	Organic	Tailing	Not specified
Native species					
Trees	0.002	0.024	0.022	-	0.000
Shrubs	-	0.009	0.022	-	0.000
Annuals	0.007	-	-	0.006	0.001
Cultured species					
Cereals	0.001	0.031	-	-	0.000
Fruits	0.002	-	-	-	0.004
Vegetables	0.008	0.000	-	-	0.001
Root crops	0.002	0.021	-	1.9	0.000
Forage	0.008	0.000	-	0.004	0.002

Overall observed geometric mean = 0.004.
1Extrapolated value based on other elements (Th and 210Pb).

stimulating effect of uranium on plant growth, such as that reported more recently by Morishima et al (1976), who observed a response to soil uranium in radishes that is similar to that of other nutrients. However, more recent literature suggests that despite the ubiquitous presence of uranium in plants, it is unlikely to be an essential micronutrient (Venugopal and Luckey 1978; Kabata-Pendias and Pendias 1984). Furthermore, a possible biochemical role for uranium has not been identified.

Southam and Ehrlich (1943) proposed the term hormesis to describe the stimulation of growth caused by sub-lethal concentrations of toxic substances. Evidence of hormesis does not necessarily indicate that a compound or element is essential. For example, one potential mechanism of hormesis is the interaction of a hormetic compound with essential nutrients resulting in enhanced uptake of the latter and subsequent growth stimulation (Meyer et al 1998a). For uranium, an interaction with phosphate may be having this effect.

In a study of the effect of DU on biomass in three range grasses, Meyer et al (1998a) found a threshold response in *Aristida purpurea* and *Buchoe dactyloides* in which no change in plant biomass occurred at DU applications to the soil of up to 5000 mg uranium per kg, but at the highest applied concentration of 25000 mg uranium per kg plant biomass decreased. In contrast, *Schizachyrium scoparium* demonstrated considerable growth stimulation at uranium concentrations of 50 and 500 mg/kg. The mechanism of the effect was not clear.

4.6.5 Uranium toxicity in plants

The available literature gives conflicting information on whether uranium is toxic to plants. Toxicity has been reported at less than ten mg uranium per kg soil, whereas no toxicity has been reported at concentrations that are several orders of magnitude above this value. However, Sheppard et al (1992) commented that studies showing toxicity at very low concentrations of uranium in soil are difficult to confirm due to the lack of relevant ancillary information and uncertainty in measurements and methodology. Better-supported studies tend to show that there is no toxic effect at much higher levels (Sheppard 1989). In an extensive recent study, Sheppard et al (1992) found no significant toxic effects at concentrations below 300 mg uranium per kg soil, and commented that in areas where such uranium concentrations occur it is likely that other contaminants, such as arsenic, are more likely to cause toxic effects. In their studies, they found species differences in thresholds for toxicity, with four crops tested affected by 1000 mg uranium per kg in soil, whereas beans (*Brassica rapa*) were not. They noted that uranium is not very toxic with respect to germination, but may have an effect in reducing phosphatase activity across a range of soil types. Further conflicting evidence on the toxicity of DU to plants is cited in Erickson et al (1990), suggesting that DU is toxic at a soil concentration of 50 mg/kg (Hanson 1974).

In a more recent study, Jain and Aery (1997) showed that uranium was toxic at high concentrations in irrigation water to wheat, leading to a significant detrimental effect on a number of metabolic growth parameters. Toxic effects gradually increased as uranium concentration in the water increased from one to 625 micrograms per litre. They also observed a reduction in uranium translocation in the plant with increasing uranium concentrations, which was thought to be due to reduced metabolic activity in roots.

4.6.6 Soil adhesion on plant surfaces

Because concentrations of many metals in the fine clay size fractions of surface soil often considerably exceed those in vegetation, a small amount of adherent soil in plant surfaces can constitute a significant proportion of ingested metal if the plant is eaten. The lower the extent of root uptake, the greater the potential importance of surface contamination by adherent soil. Because uranium concentrations in plants are usually at least two orders of magnitude lower than those on soil, adhesion of soil to plant surfaces can constitute a significant proportion of the uranium measured on plants sampled from the environment, especially if they have not been treated to remove adherent soil. Sheppard and Evenden (1988a,b) attributed very high CR values for root crops in mine tailing areas to direct soil contamination.

In desert and other environments, uranium determined in vegetation and plant litter samples has been largely attributed to particulate contamination of samples due to soil adhesion.

4.6.7 Studies on the environmental behaviour of DU

There are very few data on the rates of contamination of plants by DU. In experimental studies with three grass species typical of arid ecosystems, Meyer and McLendon (1997) reported that DU concentrations in soil as high as 25000 mg/kg were not toxic to plants. Elevated levels of DU have been reported at sites where military testing has been conducted and observations with respect to their impact on plants are discussed further in Section 5.3.

4.6.8 Summary

The plant uptake of uranium is generally low compared with many elements, but is higher than that of other transuranic radionuclides such as Pu. There are notable exceptions: some plants can accumulate high uranium concentrations in their roots. Concentration (CR) values are highly variable and decrease with increasing soil uranium concentration. Thus CR values derived from uncontaminated sites cannot automatically be used for highly contaminated areas. The effects of chemical speciation of uranium on CR values and synergistic effects of other major and trace elements on uranium uptake are poorly understood. Generalised CR values for food groups represent a simplistic model of root uptake of uranium by plants. Site-specific values are always preferable for assessments. In their absence, using conservatively high values can accommodate uncertainty due to the high variability, although the use of such conservative values directly affects the accuracy and validity of any assessment of potential harm.

4.7 Animals

Although transfer of uranium from the diet is low compared with mobile radionuclides such as radiocaesium and radioiodine, it can be higher than that of other actinides. For instance, Garten et al (1981) reported that accumulation by a range of small mammals on a contaminated floodplain biota was greater for uranium than for Th and Pu.

Exposure to animals in the environment occurs through inhalation via the lungs, ingestion via the gut or through the skin. The relative importance of each of these exposure routes depends on the physical and chemical nature of the uranium to which individual animals may be exposed.

Once circulating in the body, uranium can accumulate on bone surfaces, accumulate in the kidney and liver, or be excreted via the kidney into urine. Studies on the transfer of uranium in the environment to domestic animals are limited and, therefore, few data are available that can be used to quantify and predict the transfer of uranium to animals that are important in the human diet, especially for ruminants such as cattle, sheep and goats where data for monogastrics may not be readily transferable.

The extent of absorption via the inhalation pathway depends on the size and chemical form of the inhaled uranium, which influence the degree to which uranium penetrates the lung compartment and the extent to which it dissolves in the lung. These routes have been reviewed elsewhere and are confined to non-ruminants so will not be considered further here.

4.7.1 Absorption in the gut

Gut uptake of uranium is low, thus most ingested uranium is excreted in faeces, and could then be recycled in the environment. Direct estimates of fractional absorption in the gut are not available, but comparisons of uranium intake and excretion have been carried out in Russia and the USA. Kovalsky (1977) reported data giving fractional absorption of uranium in sheep grazing in an uncontaminated area of about 0.11 (Borovsk), and lower values in contaminated environments at Kol-Mainok and Cholpon-Ata (0.03-0.05). For lactating Holstein beef cattle, a value of 0.06 has been derived from the data of Chapman and Hammons (1963) for an uncontaminated environment. For ruminants, Cramp et al (1990) have recommended a value of 0.1, which is higher than that for monogastrics. Values calculated for pigs and chickens by Cramp et al (1990) on the basis of reported data give a figure in the region of 0.01-0.02.

4.7.2 Toxicity

In a study of the toxicity of uranium to cattle, Garner (1963) reports that in two cows receiving four mg per day, deterioration in general health over a period of two weeks with a concomitant decrease in milk yield was noted. However, despite continued administration of uranium, there was a gradual return to an apparently normal state thereafter. At the Yuma Proving Ground in the USA, slightly elevated concentrations of uranium were observed

Table 9. Reported review values of transfer coefficients for uranium to animal products (Cramp et al 1990)

Species/product	Transfer coefficient (day/kg)	
	Expected value	Range
Cow milk	4.0×10^{-4}	$7.3 \times 10^{-5} – 6.1 \times 10^{-4}$
Beef	3.4×10^{-4}	
Pork	6.2×10^{-2}	
Poultry	1.2	$3.0 \times 10^{-1} – 1.2$
Egg	1.0	

in all ecosystem components. However, autopsy of animals showed their kidney uranium content to be below threshold values in all species, except for Kangaroo rats in which histopathology indicated possible damage to kidney tissue (Ebinger et al 1996). The consumption of dust, which had become adhered to foliage, was the most important exposure pathway for animals living in these sites.

4.7.3 Body distribution

There is a lack of experimental studies with domestic animals that provide mechanistic information on the rates of accumulation and loss from body tissues.
Measurements of uranium contamination in ruminants have shown that uranium accumulates primarily in bone, in terms of the total content in the body, but also in muscle due to its importance as a proportion of total body mass (note that muscle uranium concentrations are generally low). Compared with other body tissues, high concentrations have been reported in the kidney, liver and tracheobronchial lymph nodes. In contaminated sites, uranium has also been found in the pelt of small mammals (eg Hanson and Miera 1976) and in gut contents.

Reported concentrations of uranium and DU in animal tissues will be affected by the recent diet of the animal, and how rapidly the uranium is excreted from each tissue after deposition. Tissues in which uranium has a relatively short biological half-life (such as the kidney) will accumulate and lose uranium faster than other tissues. In contrast, biological half-lives in bone are generally longer than those in soft tissues (one to several years; Linsalata (1994)).

4.7.4 Quantification of transfer from plants to animals

The transfer of heavy metals or radionuclides from plants to animals is often quantified using a concentration ratio (CR) between the animal (or a specified tissue) (eg mg/kg or Bq/kg fresh weight) and the vegetation that it ingests (eg mg/kg or Bq/kg dry weight). The CR is often used for uranium in extensive ecosystems. In contrast, the transfer of radionuclides, including uranium, in intensive agricultural systems is more frequently quantified using the transfer coefficient (day/kg), defined as the equilibrium ratio between the activity concentration in the specified animal tissue (Bq/kg fresh weight) and the daily intake of the radionuclide by the animal (Bq/day).

There have been a number of compilations of transfer data for uranium to agricultural animals that are shown in Table 9. The values given in the table are based on only a few measurements. The highest transfer coefficients are recorded for eggs and poultry.

The transfer coefficients used by the NRPB within their generalised derived limit (GDL) assessments are shown in Table 10 (NRPB 2000).

4.7.4.1 Environmental measurements of uranium contamination in animals

Measurements of uranium in tissues of animals grazing in uranium-contaminated areas have been reported to be higher than those in control areas. In one of the few field studies comparing uranium concentrations in domestic species, Linsalata et al (1991) reported that uranium concentrations in muscle decreased in the order: chicken>beef cattle>pig. Lapham et al (1989) reported significantly higher uranium concentrations in cattle kidney and liver, but not in muscle in cattle grazing in an area contaminated by uranium mining. Smith and Black (1985) reported slightly elevated levels in cattle grazing near the Rocky Flats plant in Colorado.

Few measurements of uranium in wild animals have been made, but those compiled do not report significant accumulation in tissues (eg Clulow et al 1996), although they are measurable, and often elevated in whole animal samples at contaminated sites. Concentration ratios have been reported for caribou muscle compared with lichen of 0.01-0.16 for uranium, which can be compared with 0.06-0.25 for ^{226}Ra, 0.01-0.02 for ^{210}Pb, 0.06-0.26 for ^{210}Po and 2.60-3.70 for ^{137}Cs (Thomas and Gates 1999).

In a study by Thomas (2000a), the behaviour of uranium, ^{226}Ra, ^{210}Pb and ^{210}Po in a bog and a pine habitat were compared. Deer mice had higher uranium concentrations than meadow voles. The ratio of uranium/^{226}Ra was higher in birds than in small mammals. When considering transfer, Thomas (2000b) found that CR values were lower at

Table 10. Transfer coefficients used for uranium in the GDL assessments by the NRPB

Species/product	Transfer coefficient (day/kg)
Cow milk	6.0×10^{-4}
Beef	2.0×10^{-4}
Sheep meat	2.0×10^{-3}
Cow offal	2.0×10^{-4}
Sheep offal	2.0×10^{-3}
Milk products	6.6×10^{-3}

contaminated sites than at 'natural background' level sites. He attributed the difference to association of radionuclides with particles in dusts, which were presumed to have a low bioavailability. Thomas suggested that soil ingestion is a major dietary source of the radionuclides, since activity concentrations are usually higher in soil than in vegetation and soil-burrowing animals can potentially ingest large amounts of soil.

4.7.5 Soil-associated uranium intake

Animals generally eat more soil than humans; herbivores eat soil adhered to vegetation and soil associated with root tissues. Grazing leads to more soil ingestion than feeding with cut forage. The ingestion of contaminated soil by grazing animals varies with stocking rate, herbage intake rates, pasture conditions, forage type and season. Higher quantities of soil are likely to be consumed when there is a low herbage biomass (especially in winter) and a high stocking rate. Large quantities of soil can be consumed from selected areas, which often have a high salt concentration, indicating that the soil is supplying a dietary need.

Because concentrations of many metals in surface soil often considerably exceed those in vegetation, a small amount of adherent soil on plant surfaces can constitute a significant proportion of ingested metal if the plant is eaten. The lower the extent of root uptake, the greater the potential importance of surface contamination by adherent soil.

In a recent review, WHO (2001) stated that typical soil ingestion values for cattle are about 500 g per day and are 60 g and 500 g per day for sheep and pigs, respectively, on the basis of live weight. The NRPB (2000) assumes that a sheep ingests 0.3 kg/day of soil for a daily foodstuff intake of 1.5 kg; for cattle the soil intake is assumed to be 0.52 kg/day and a herbage intake of 13 kg/day.

Isotopic ratios in farm animal tissues were shown to resemble closely those in soils over which the animals forage (Linsalata et al 1991). This indicated the importance of soil ingestion as a source of ingested uranium.

For animals, ingestion of soil may be a major potential exposure route for uranium and DU, and is likely to vary considerably due to the factors mentioned above. In addition to ingestion, soil-associated uranium and DU may reside within hair, fur or wool.

The relative bioavailability of uranium or DU ingested via soil consumption may differ from that in herbage. However, there are no data available to indicate the relative bioavailability of the different sources. In subsistence communities most fodder is grown locally. For some subsistence communities, available land for private production is of poor quality and, under these conditions, particularly in winter, soil consumption may be high. Herbivores ingesting soil whilst browsing may ingest particulate DU present in upper soil layers, especially the root mat and DU adhered to vegetation surfaces. DU intake will obviously be lower if domestic animals are supplied with fodder grown outside the contaminated area.

4.7.6 Summary

There are very few data quantifying the transfer of both uranium and particularly DU for domestic animal species in both agricultural and extensive ecosystem habitats. Due to the low uptake of uranium by plants, adherent soil on plants, which is ingested by animals, may constitute a major source of uranium. No data are available on the bioavailability of soil-associated uranium or DU for gut uptake

4.8 Humans

Humans may become exposed to uranium from either natural or man-made sources, whilst exposure to DU only results from anthropogenic activities. Possible routes of exposure are similar to those discussed previously for other mammals and include inhalation, ingestion, dermal absorption and direct introduction into the body via injury or insult. Because uranium is radioactive it is also possible for humans to be externally exposed to radiation. Exposures to uranium and DU via these routes have been extensively reviewed in previous studies (eg ATSDR 1999; UNEP 2000) and exposures during military conflict were discussed in Part I of the report. For this reason, exposures via these routes are only discussed in terms of their dependence on

environmental pathways (Sections 4.1 to 4.7 above) and in the context of data relating to potential source terms. Physiological factors controlling exposure and uptake of DU along with toxicological and radiological implications are discussed in Chapter 1 and Appendix 1 of this report and in Part I of the report, and are therefore also not discussed in detail in this section unless they are directly relevant.

The relative importance of each of the exposure pathways discussed below is very dependent upon the source of the DU (see Section 2). For example, during or immediately after its release into the environment the most important factor influencing exposure is the amount of DU metal that is converted into dust (both respirable dust that may be inhaled and non-respirable dust that may become ingested via a number of routes such as inadvertent soil ingestion). Over longer timescales other routes of exposure less related to the direct or indirect ingestion of dusts may become dominant. For example, dusts will weather or metallic fragments corrode, producing secondary products that may be taken up into the food chain and ingested. In the context of the military use of DU, the relative importance of inhalation and ingestion depends upon the military tactics being employed and upon the prevalence of hardened targets on the battlefield. The use of DU against a foe with a poor standard of protective armour (eg infantry or buildings) would be expected to produce a lower concentration of respirable dusts compared with an attack on a heavily armoured target such as a modern main battle tank. Similarly, strafing attacks that often result in a poor target hit rate when compared with tank-tank battles would be expected to produce a much lower proportion of respirable material per strike.

Although dermal sorption through intact skin potentially represents a route of human exposure, there is no evidence to suggest that the magnitude of this route of exposure is likely to result in any health impact when DU has been used in military conflict. This is because DU combustion products and residual fragments of DU-Ti alloy are significantly less soluble and/or present at significantly lower concentrations than those in situations where dermal sorption has been shown to occur in animals (see Chapter 1 on the chemical toxicity of DU). However, despite this observation, a precautionary approach would be to use personal protective equipment particularly when handling potentially contaminated dusts and soils from the immediate vicinity of penetrator strikes (eg within 20 to 30 cm).

Without comparative data from different types of conflict, or a sufficiently robust model, it is difficult to compare the relative levels of exposures following these various military uses of DU. However, data collected to date (eg IAEA Workshop 2001; Priest and Thirlwall, personal communication; UNEP 2001) from the Kosovo conflict in which relatively large numbers of 30 mm penetrators were used in strafing attacks suggest that overall levels of DU contamination of the near-surface environment immediately following the conflict were comparatively low when compared with those observed in military proving grounds in the USA (eg AEPI 1995), where intensive use of DU has occurred over a number of years. This statement must, however, be qualified as less than 25% of the total number of DU penetrators have been located, contamination levels within 20 cm of penetrator strikes may be very high, and contamination of the subsurface environment and subsequent migration into groundwater and/or surface ecosystems may take tens of years to become manifest.

4.8.1 Air

As is the case for animals, humans may inhale or ingest particulate DU. During a conflict the dominant mechanism responsible for the introduction of DU into the atmosphere is that of combustion and impact energy. After a number of hours initially suspended material will settle out and secondary resuspension will become the dominant factor leading to the inhalation or atmospheric transport of DU. These issues have been discussed previously in Part I of the report. Over the longer term, particulate DU will be removed from the Earth's surface leading to a steady decrease in the potential for resuspension. For example, high rainfall and/or weathering will encourage the removal and dispersal of DU dusts and small fragments from the battlefield and into deeper soil profiles and/or surface drainage networks. The potential for resuspension of DU dusts has been modelled using available data for a generic situation in Annexe B and illustrates that the relative importance of this exposure pathway will decrease with time. Even using conservative assumptions, levels of DU in resuspended air are estimated to be in the order of 10^{-8} g/m^3 reducing to 10^{-9} g/m^3 over a period of ten years, and these represent concentrations over a million times less than those used in Part I of the report to estimate potential effects on the health of some exposed soldiers. As described in Section 4.1 the natural background air concentration of uranium in air is in the order of 10^{-10} to 10^{-9} g/m^3. Estimated lifetime intakes of DU from the inhalation of resuspended material are in the order of 0.1 mg (central estimate) and three mg (worst-case estimate), and these compare with that expected from the inhalation of uncontaminated air (0.23 mg). Given these factors it is unlikely that those casually entering an area of conflict after a period of a week or two will be exposed to anywhere near the level for those present in the immediate vicinity during the aftermath of a penetrator strike. This is provided of course that they do not engage in a specific activity that would significantly promote the resuspension of any DU contamination (eg entering heavily contaminated struck vehicles).

The processes described above would normally be expected to result in increased dispersal of DU; however, the unusually dense nature of DU may lead to secondary concentrations of such particles in suitably favourable niche environments where less dense materials may be preferentially removed (eg in areas of rapid water flow or exceptionally windy conditions).

4.8.2 Soil

Humans, particularly the young, may directly ingest soil or domestic dust inadvertently, deliberately or habitually, and these modes of exposure have been considered to be of particular significance where other sources of exposure are well controlled. Factors of particular relevance are the quantity of soil or domestic dust ingested, the measured concentration of DU in the medium, the accessibility of the soil, and the availability and rate of adsorption of DU in such soils in comparison with materials that have been used in the assessment of toxicity. In Annexe C potential scenarios for the ingestion of soil are developed and resultant exposures and radiological doses calculated; however, in doing this it is very difficult to take into account the sporadic nature of such activities and every potential scenario. Whilst some will question the high levels of soil ingestion used in Annexe C, it should be considered that these consumption values - which are increasingly being substantiated during the course of risk assessment methodologies associated with contaminated land, and human nutrition amongst tribal and ethnic communities - may be underestimates because the relatively high density of DU and its combustion products will tend to increase exposures reliant on volume rather than mass.

Results of calculations indicate that exposures due to inadvertent soil ingestion are unlikely to be of concern from the perspective of human health, even if it is assumed that such exposures occur in an area in which DU concentrations are in the order of 100 mg/kg. These concentrations have only been recorded within about 20 cm of penetrator strikes in Kosovo, but evidence from DU testing sites would, however, suggest that such concentrations might be found over larger areas under some conflict scenarios (eg following intensive tank battles).

Of more concern from an exposure and human health perspective are exposures due to both one-off deliberate consumption and habitual consumption of contaminated soil. This is particularly the case should DU be used in areas inhabited by disadvantaged or tribal communities in which practices such as geophagy (eating of soil) are common. Care also needs to be exercised to establish the likelihood of such practices in areas such as the Balkans and Iraq, particularly as personal communications with aid workers suggest that geophagy is practised in these regions. Given the lack of evidence for widespread DU contamination from UNEP studies (UNEP 2001), it would seem reasonable to assume that consumption of contaminated soils on a regular basis is unlikely, and hence that calculated exposures and radiological doses received under the scenarios assumed for a geophagic individual are overly conservative (eg radiation dose = 15 to 50 millisieverts per year and chemical exposure = 9.5 to 31 g uranium per year). However, calculations show that chemical exposures, and potentially also radiological doses, may be exceeded following a relatively small number of occasional deliberate events, which are probably more realistic issues given the nature of sporadic contamination observed in Kosovo in which concentrations of up to 18 g uranium per kg soil have been measured at strike sites (UNEP 2001). Such exposures may be readily limited by relatively unsophisticated methodologies such as clearly marking strike sites as being out of bounds (although this may be more of an attractant to children and young adults), and the careful physical removal of soils and dusts from the immediate vicinity of strike sites.

Doses and exposures calculated in Annexe C are based on an assumption that all of the DU is bioavailable, which is probably a highly conservative assumption. However, few data exist on the bioavailability of DU-Ti alloys and associated combustion products in the human gastrointestinal tract (WHO 2001). Given the potential for elevated exposures via the ingestion of soils and dusts, this area represents a significant knowledge gap in current studies. The general assumption that uranium in contaminated soils is likely to be of low solubility has already been questioned by Elless et al (1997), who clearly demonstrated that anthropogenic uranium may be significantly more soluble (up to 40% of the total uranium being soluble) in stomach fluid simulants than naturally occurring soil uranium. However, as stated above, results for dusts produced from the impact of DU-Ti alloys may be significantly different.

4.8.3 Water

The main concern for water resources in the case of DU is exposure through direct ingestion, particularly as drinking water is often the main contributor of uranium in the human diet (eg ATSDR 1999). Other forms of exposure through, for example, the ingestion of fish derived from contaminated water resources and direct absorption through skin contact at levels likely to be encountered after the military use of DU (based on data from Kosovo) are considered to be minimal. Under significantly higher levels of contamination, such as those present in the immediate vicinity of uranium mining and milling sites, the bioconcentration of uranium in the aquatic food chain has been noted (Clulow et al 1998), although potential intakes from the ingestion of fish were relatively low (2.3 mg/year) and comparable to intakes from other uncontaminated sources.

WHO has recommended a guideline of two microgram per litre for drinking water (WHO 1998a), based on the potential for negative impacts on kidney function. Implicit assumptions used in the derivation of this limit were that it should be protective across all members of the population, including potentially sensitive subgroups such as the infirm, children and aged, and that the exposure from drinking water should not exceed ten percent of the total exposure limit. It is therefore unsurprising that this limit is exceeded in many water supplies (by up to a factor of 1000) without apparently serious negative impacts (WHO 2001), although a dose-dependent relationship between levels of uranium in drinking water and indicators of kidney dysfunction have been observed in some studies. These studies and other issues related to the potential toxic effects of uranium and associated epidemiological data are discussed in Chapter 1 and Appendix 1. As discussed in Section 5 and Annexe F, contamination of water resources by soluble DU would be unlikely to be immediately measurable even in wells within ten m of a strike site, unless a penetrator became directly lodged in the well or borehole.

4.8.4 Other foodstuffs

Exposure to DU through the ingestion of foodstuffs is likely to be limited because of the relatively low bioconcentration of uranium into animals and plants that may be used as foods (see Sections 4.6 and 4.7 above). Other studies of exposure to uranium (eg ATSDR 1999; WHO 2001) highlight the potential for exposure via adhered contaminated soils and dusts when eating unprepared foods or when food hygiene is poor. Similarly, the drying of foods directly on potentially contaminated soils is a possible route of exposure. Whilst no specific international recommendations or guidelines exist governing the concentration of either uranium or DU in the UK, the NRPB has produced a series of generalised derived limits for the presence of uranium in foodstuffs (NRPB 2000). The derivation of these values and an analogous set of guidelines based on the generic tolerable daily intakes derived in WHO (2001) are discussed in the following section. Values derived from this exercise emphasise the importance of monitoring drinking water and milk in areas in which DU has been used, both from the context of radiological and chemical toxicity, and emphasise that derived limits based on chemical toxicity are also protective against potential radiological impacts. Levels of uranium in plants and animal products would not be expected to limit human use of such vegetation for dietary reasons, provided that total intakes do not exceed those generic levels derived in Section 5.

4.8.5 Summary

Exposure of humans to DU may occur through three principal pathways: inhalation, ingestion and dermal absorption. As has been discussed above, in the case of animals, the relative importance of each of these exposure routes depends on the physical and chemical nature of the uranium to which individuals may be exposed. Exposure to naturally occurring uranium can occur via the consumption of a wide range of foodstuffs, all of which contain uranium to some extent, but in many systems is likely to be dominated by the inhalation and ingestion of dusts and soil (either directly or through the ingestion of soil or dusts adhered to the foliage of plants) and drinking water. However, the dominant pathways in the case of DU are dependent upon the nature of the contaminative event and the time elapsed between the release of DU into the environment and the extent of exposure. For example, during a conflict, exposure to those in the immediate vicinity of penetrator sites will be dominated by inhalation, whilst exposure to those living in the vicinity of a combat zone 50 years later may be dominated by ingestion, as the DU contamination has settled out from the air, and DU has been solubilised from buried penetrators and become increasingly evenly dispersed amongst soil, plants and drinking water.

5. Frameworks for the assessment of the environmental impact of DU

Contamination resulting from the use of DU can be assessed by either:

(1) comparing measured levels of contamination with established guideline or screening values, or
(2) applying generic models whereby exposures and effects on receptors such as humans can be estimated for generic exposure scenarios, or
(3) applying site-specific models whereby exposures and effects on receptors such as humans can be estimated for specific exposure scenarios.

All of these procedures have distinct roles in the assessment of the potentially harmful effects of contamination resulting from DU and may be applied sequentially or in parallel depending on the availability of data and the potential impact. The use of guidelines or screening values is indispensable in preliminary assessments and may be necessary to comply with legal requirements in some countries or situations. However, these methods, based on pessimistic scenarios, tend to be conservative and in the case of DU guidelines may be less well established than for other more common potentially toxic elements such as lead. Because of the lack of such definitive guidelines, and a lack of site-specific data, generic models have generally been employed to date in the study of potential DU contamination from the Gulf War or Kosovo conflict (eg Fetter and von Hippel 1999; UNEP 2000). However, as our understanding of the use of DU munitions increases, there is a clear role emerging for the use of more detailed site-specific models, particularly in detailed investigations, to provide additional tools to

minimise knowledge gaps and uncertainties in the assessment of exposures and risks. These issues are highlighted in the following sections, which focus upon examples of three general methodologies - monitoring, numerical modelling and the derivation of generalised derived limits - and illustrate the uncertainties associated with a more generic approach to environmental assessments.

5.1 Monitoring

Environmental monitoring should represent the easiest way to ensure protection against exposure to potentially harmful substances that may have been released into the environment. Whilst this is generally true where suitably accurate and precise methods of chemical analysis exist, and conservative guidelines for particular receptors have been established, situations exists where this is not the case. For example, where exposure to a number of potentially harmful substances occurs simultaneously at levels below which any one individual substance would be expected to cause harm, or where the use of overly conservative assumptions cannot be justified on the basis of health-related evidence. Such cases are increasingly being highlighted when considering, for example, ecosystem exposures, particularly where guidelines have not been established or complex interspecies interactions are poorly understood.

In the context of monitoring for both uranium and DU, the development in the late 1980s of inductively coupled plasma mass spectrometry (ICP-MS), coupled with continuing improvements in alpha spectrometry, neutron activation analysis and X-ray fluorescence spectrometry (XRFS), have meant that affordable, suitably accurate and precise techniques exist for the measurement of uranium and DU in many environmental matrices (Ivanovich and Harmon 1982; IAEA 1989; Gill 1997; Toole et al 1997). The use of accelerator mass spectrometry has also proved useful in the identification of ^{236}U in environmental samples (Marsden et al 2001). Chemical analysis by ICP-MS and XRFS have the added advantage in allowing the simultaneous monitoring of a wide range of other environmental contaminants that may result from conflict, or from natural or anthropogenic sources, and monitoring for DU contamination can be incorporated into ongoing national monitoring strategies related to food and water quality. The use of these and other associated field techniques for the identification of gross DU contamination are reviewed in UNEP (2001).

In the context of ecosystem monitoring, the use of sensitive sentinel organisms is increasingly being suggested as an alternative technique to substance-specific chemical analysis for monitoring harm to the natural environment. In the case of uranium, and hence DU, *in-vivo* and *in-vitro* testing on the clam *Corbicula fluminea*, the worm *Eisenia fetida andrei* and the teleost fish *Brachydanio rerio* has been undertaken to establish if such species may be used as sentinel organisms (Labrot et al 1996).

A number of studies using conventional analytical techniques have also been undertaken to investigate the levels of atmospherically distributed uranium and DU in mosses, lichens and tree bark (eg Ma et al 2001; UNEP 2001). With further development, and timely application in areas of conflict, such studies are likely to improve our understanding of the distribution and scale of particulate DU released from both military conflicts and the testing of DU munitions. The increasing use of analytical instruments capable of measuring uranium isotopes at extremely low uranium concentrations is resulting in an increasingly large amount of reliable data regarding the abundance and distribution of natural and anthropogenically introduced uranium. However, the interpretation of such data requires care as ratios of naturally occurring uranium isotopes vary due to entirely natural process and other sources of anthropogenic DU exist, so the presence of measurable DU concentrations should not be automatically used to imply harm.

The identification of extremely localised 'hot spots' of DU contamination associated with penetrator strikes during investigations in Kosovo by UNEP (UNEP, 2001) also highlights problems associated with the interpretation and comparison of monitoring data. Similarly, the particulate nature and high density of primary and secondary forms of DU potentially invalidate, and certainly complicate, the derivation and interpretation of 'average DU concentrations', particularly at low levels.

5.2 Numerical modelling

Models may be considered to be idealised and simplified representations of complex systems. In the context of this discussion, models provide opportunities for drawing quantitative or semi-quantitative conclusions regarding transfers of substances between various environmental compartments and the likely exposures of specified receptors such as man or groundwater. Such models underpin the assessment of potential risks associated with the release of potentially harmful substances (eg Ferguson et al 1998; NRPB 2000; WHO 2000). The degree and numerical nature of the modelling undertaken is usually proportional to the complexity of the system under study and the accuracy with which an assessment of exposure is required. For example, a more accurate assessment may be required if exposure could result in a particularly high degree of harm or where precautionary monitoring is difficult or impossible. Similarly, a more complex model, or series of sub-models, is required to model accurately a complex system. A major limitation to the resultant accuracy, and hence applicability, of any environmental model is the degree of uncertainty associated with the variables

required by the model (eg in the case of environmental exposures to uranium the wide range in K_d associated with broadly similar soils is one factor limiting the accuracy of predicting the transport of uranium into groundwater). The ever-present issue of uncertainty and heterogeneity in natural systems has in some cases led to the development of probabilistic models, an example of which is used in this section to investigate the likelihood of a groundwater source becoming contaminated with DU.

5.2.1. Derivation of generalised derived limits

The principal potential exposure routes by which humans may receive either a radiation dose or chemical exposure in the terrestrial, freshwater or marine environments are broadly similar and are summarised in Figure 8. These exposure routes or pathways form the focus of a wide range of exposure assessment models that have been developed to underpin risk assessment methodologies in both the chemical (Ferguson et al 1998) and nuclear industries (IAEA 2001). Such models may be used either to estimate exposures given a defined scenario, or can be inverted to estimate contaminant concentrations in environmental materials that result in a reference dose or guideline value being exceeded by the receptor (eg a human). In this section the latter method is used to develop generalised derived limits (GDLs) for exposure to uranium for a radiological dose of one mSv per annum or, in the case of chemical toxicity, the recommended tolerable daily intake (see Chapter 1).

5.2.1.1 Derivation of radiological Generalised Derived Limits

5.2.1.1.1 Methodology

GDLs have been developed within the radiation protection community to provide convenient reference levels against which the results of environmental monitoring can be compared (NRPB 2000). They are based upon the radiological exposure of humans via a number of well-defined potential pathways and principles. For example, while present in the air, DU may give exposures by:

- external irradiation by photons and electrons emitted as a result of the radioactive decay process
- internal irradiation following their inhalation.

The processes of deposition onto underlying surfaces will gradually remove DU from the air. The deposition of DU onto the ground, and onto other surfaces, leads to further transfer in the terrestrial environment where humans can continue to be exposed to DU. A number of exposure routes may occur here:

- deposited DU may still be available for inhalation as a result of resuspension, caused by wind-driven or man-made disturbance
- radioactive decay of deposited DU will also lead to external exposure from photons and electrons
- deposition onto vegetation and soils leads to the transfer of radionuclides into human foodstuffs and into water, the consumption of which will lead to internal exposure
- there may be inadvertent ingestion of contaminated soils.

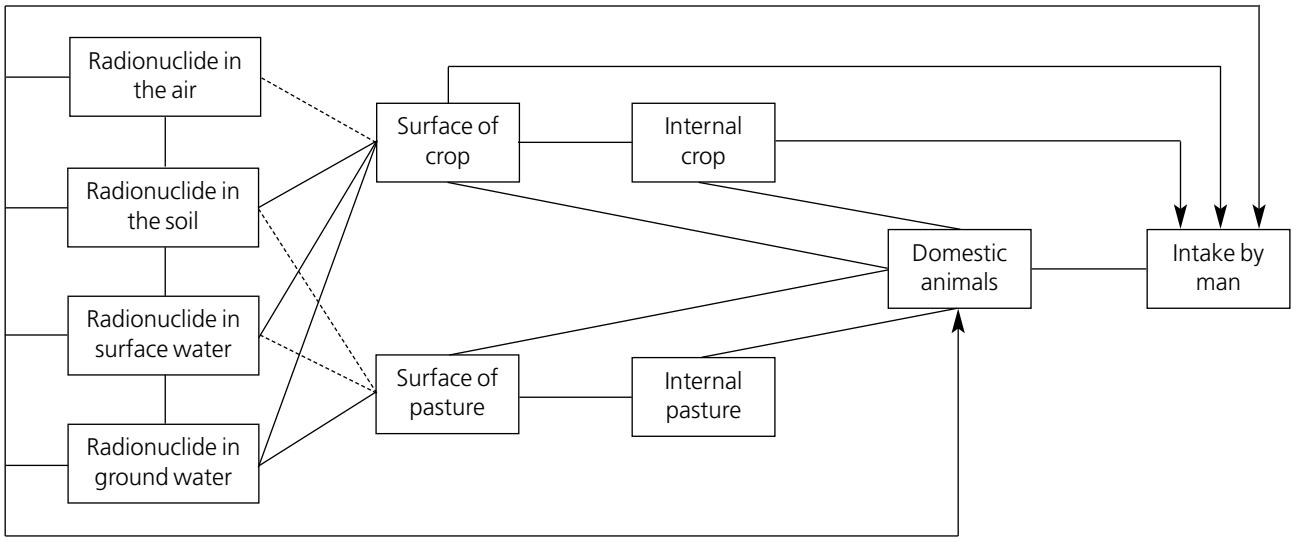
The relative importance of these pathways depends on the form of the radionuclide and the nature of the surface onto which the deposition occurs. For example, ingestion pathways may be less important than external and inhalation pathways in an urban area compared with a rural area. The exposures of people can be assessed in terms of individual and collective (or population) doses. For this, appropriate dosimetric models and habit data are also required, in addition to models that predict atmospheric dispersion and environmental transfer. In the case of DU, removal by radioactive decay can be ignored because of its long half-life.

GDLs relate to the annual effective dose limit for members of the public in the UK of one mSv, and as such GDLs relate only to possible increases in radioactivity resulting from human activities, and do not include the contribution to dose, possibly larger, from natural background radiation. As GDLs relate only to incremental concentrations of radioactivity resulting from human activities, and not to the total concentration measured, an estimate of the ambient levels in the area of interest should be obtained, to subtract from any measured concentrations of radioactivity before comparison with the appropriate GDL.

Being generic, GDLs are calculated using deliberately cautious assumptions, and are based on the assumption that the level of environmental contamination is uniform over a year. For application in the UK, it is recommended that whenever a measured environmental concentration exceeds about 10% of the concentration limit implied by the GDL, then the doses should be examined more closely. Any fuller examination would take account of site-specific factors and the length of time the measured level is likely to be maintained. The NRPB (NRPB 2000) has published GDLs for ^{234}U, ^{235}U and ^{238}U. In this section, and in Annexe D, we describe the philosophy of GDLs and the assumptions underlying the calculation of GDLs for well-mixed soil and aquatic pathways. The GDLs for ^{238}U for these pathways are given and, for the GDL for well-mixed soil, the relative significance of the exposure pathways is indicated.

GDLs are calculated using effective dose as defined in ICRP Publication 60 (one millisievert per annum) (ICRP 1991). The values for dose coefficients from inhalation

Figure 8. Exposure pathways commonly considered during environmental modelling of exposure to radioactivity



and ingestion assumed for ^{238}U are given in Table 1 in Annexe D. The GDLs are based on the dose to the most restrictive age group, taking into account variations in dose coefficients and dietary and other habits with age. The age groups considered are infants (one year old), children (ten years old) and adults (assumed to be 20 years old). In addition, for GDLs in milk or where the ingestion of milk could be the dominant pathway, calculations are also performed for infants on a milk diet in the first year of life based on dose coefficients for a three month old.

5.2.1.1.2 Results and discussion
GDLs for uranium are presented in Table 11. The relative importance of pathways for well-mixed soil GDLs shows that the dominant exposure pathway associated with DU contamination of soil is the ingestion of food. Other less important exposure pathways are inadvertent ingestion and inhalation of soil, although these are of greater importance for infants and children (see also Annexe C). Comparison of individual GDLs indicate the sensitivity of air, drinking water and milk to contamination from DU. In all of these cases the critical group is children and/or infants (including those in the first year of life).

5.2.1.2 Derivation of chemical Generalised Derived Limits

5.2.1.2.1 Methodology
As described above, GDLs have been developed for exposures to radioactivity. However, in the absence of guidelines related to acceptable concentrations of uranium or DU in foods and various environmental materials (with the exception of drinking water), it was considered useful to use a similar approach to estimate reference levels of uranium in various environmental materials which could result in excess human exposure to uranium from the perspective of the tolerable daily intake for chemical toxicity. This has been undertaken by simply extrapolating the methodologies used to determine GDLs; the methods are described in Annexe E. For the purposes of this work the term general derived limit chemical (GDLC) is used to differentiate chemical GDLs from those calculated for radiological purposes.

5.2.1.2.2 Results and discussion
GDLCs for uranium are presented in Table 12. The relative importance of pathways for well-mixed soil GDLCs are similar to those for GDLs derived on a radiological basis and show that the dominant exposure pathway associated with DU contamination of soil is the ingestion of plant and animal products (Figure 1, Annexe E). In the case of soils, GDLCs for infants are close to the natural background concentration of uranium in UK soils of 0.1 to 2 mg/kg (see Section 1.2) and are considerably more restrictive than the equivalent GDL. Other less important exposure pathways from soil are inadvertent ingestion and inhalation of soil, although these are of greater importance for infants and children (see also Annexe C).

Comparison of individual GDLCs indicates the sensitivity of air, drinking water and milk to contamination from DU. In all of these cases the critical group is children and/or infants (including those in the first year of life). GDLCs for air are three orders of magnitude above natural concentrations of uranium in air and are generally also above estimated concentrations of DU due to resuspension (Annexe B). GDLCs for drinking water are higher than guidelines produced by WHO (WHO 1998a) because in the calculation of GDLCs it was assumed that 100% of the tolerable daily intake could be derived from drinking water.
Where GDLCs relate to exposure from a single environmental exposure route, it is important to note that in practice people will be exposed to a variety of

Table 11. GDLs for ^{238}U (Bq/kg)[a] (NRPB 2000)

	GDL	Critical group[1]
Single		
Inhalation of air	0.05	children aged 10 years
Locally grown fruit	200	infants aged one year
Potatoes and root vegetables	200	children aged 10 years
Green and other locally grown vegetables	300	adults
Cereals	200	children aged 10 years
Cattle meat	500	children aged 10 years
Sheep meat	900	adults
Offal[2]	1000	adults
Milk	8	infants under one year on all milk diet
Milk products	200	infants aged one year
Marine fish[3]	200	adults
Crustaceans[3]	1000	adults
Molluscs[3]	1000	adults
Drinking water	30	infants aged one year
Freshwater fish[3]	1000	adults
Multiple		
Well-mixed soil[4]	20000	infants aged one year
Freshwater sediments[4]	400000	children aged 10 years
Marine sediments[4]	100000	adults
Fresh water[5]	20	adults
Sea-washed pasture[4]	20000	infants under one year on all milk diet

[1]The GDLs apply to uniform conditions over a year and are based on the limiting age group. Unless stated otherwise, GDLs for food products are expressed as fresh mass.
[2]Offal refers to cow liver and sheep liver.
[3]The GDLs for aquatic foodstuffs are for the edible fraction and are expressed as fresh mass.
[4]The GDLs are expressed as dry mass.[5]The GDLs for fresh water include activity in the dissolved and suspended fractions.

materials/pathways, and hence in comparing these levels against environmental measurements it is necessary to take into account all possible sources of contamination to ensure that the toxicity limit is not exceeded.

5.2.2. Groundwater contamination

Exposure of water to DU contamination is likely to be dominated by transfer from direct soil deposition, where firing of DU munitions occurs over land, due to the small surface area that freshwater generally covers. The transfer of uranium from the soil, or regolith, will be controlled by physical and chemical processes, which will be regulated by the climatic and geological environment in which the contamination occurs.

The nature of DU entry onto the soil surface (eg fragmentation following impact with a target) or within the soil profile (eg burial of nearly intact penetrators) will affect the rate and mode of transfer of uranium to the soil-water, surface-water and groundwater environments. Fragmentation will increase the surface area of the penetrator available to chemical and physical weathering. Small particles may be entrained in the near-ground atmosphere during dry (dusty) conditions. Overland water flow, from rainfall or snow thaw, will cause the physical movement of particulates to surface water courses, and ultimately into estuaries and near-shore environments. Physical translocation of particulate material into groundwater may occur through the regolith and within aquifers that have secondary fracture flow mechanisms. The burial of DU penetrators from a 'soft' impact with soil will lead to little fragmentation, but could potentially contaminate groundwater resources by dissolution and migration into aquifers.

The mobility of DU in the near-surface environment will be controlled by the local environment of the penetrator which may lead to corrosion and dissolution (Figure 10), and factors such as the pH of soil minerals and water,

Table 12. GDLC (in mg/m^3, mg/kg or mg/litre DU) for various exposure routes and scenarios

	Infant (one year)	Child (ten years)	Adult (20 years)	Critical group[1]
	Single	exposure	pathways	
Inhalation of air	0.001	0.001	0.002	children aged 10 years
Locally grown fruit	0.055	0.12	0.17	infants aged one year
Potatoes and root vegetables	0.043	0.062	0.098	children aged 10 years
Green and other locally grown vegetables	0.13	0.17	0.16	infants aged one year
Cereals	0.064	0.079	0.13	children aged 10 years
Cattle meat	0.19	0.20	0.29	children aged 10 years
Sheep meat	0.64	0.59	0.51	adults
Cow offal	0.70	1.2	1.3	infant aged years
Sheep offal[2]	0.70	1.2	1.3	infants aged one year
Milk	0.006	0.025	0.053	infants aged one year
Milk products	0.043	0.13	0.21	infants aged one year
Marine fish[3]	0.38	0.30	0.13	children aged 10 years
Crustaceans[3]	1.9	1.2	0.64	children aged 10 years
Molluscs[3]	1.9	1.2	0.64	children aged 10 years
Drinking water	0.007	0.017	0.021	infants aged one year
Freshwater fish[3]	1.9	1.2	0.64	children aged 10 years
	Multiple	exposure	pathways	
Well-mixed soil[4]	4.1	10.9	15.7	infants aged one year
Freshwater sediments[4]	1300	1200	5000	infants aged one year
Marine sediments[4]	1300	2000	1300	adults
Fresh water[5]	0.0071	0.015	0.016	infants aged one year
Sea-washed pasture[4]	6.5	22	36	infants aged one year

[1]The GDLCs apply to uniform conditions over a year and are based on the limiting age group. Unless stated otherwise, GDLCs for food products are expressed as fresh mass.
[2]Offal refers to cow liver and sheep liver.
[3]The GDLCs for aquatic foodstuffs are for the edible fraction and are expressed as fresh mass.
[4]The GDLCs are expressed as dry mass.
[5]The GDLCs for fresh water include activity in the dissolved and suspended fractions. Unlike GDLs, GDLCs for fresh water do not include contributions from the ingestion of irrigated vegetables (see Table E5, Annexe E).

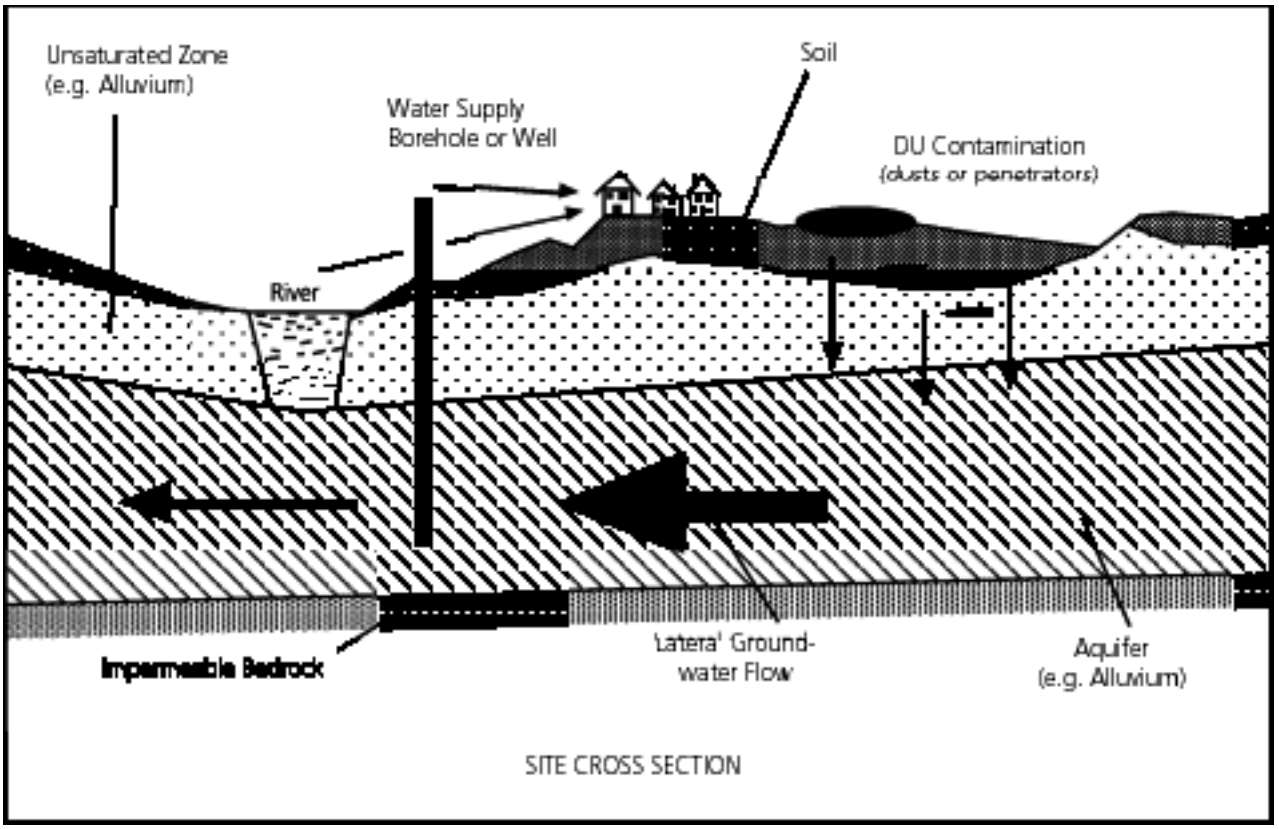
and the sorption potential of soil minerals (Section 4). Thus where soil strongly binds the uranium in secondary phases or on surfaces (eg iron oxides, clay minerals or organic carbon), its release into soil water, and translocation to groundwater, should be minimal. In deeper environments mobility and attenuation are controlled by the composition of fracture coatings and water chemistry. Where uranium is highly mobile, water resources may be more vulnerable to contamination.

The vulnerability of water to uranium contamination will be controlled by the geological conditions, soil conditions and mobility encountered. The primary factors affecting vulnerability, assuming that uranium is mobile, are the depth of the unsaturated zone (ie proximity of the contamination to the water table) and the infiltration rate of recharge. For example, the vulnerability of water resources hosted in river gravels may be high due their proximity to the surface, whilst the vulnerability of those obtained from deeper, possibly confined, aquifers will be lower.

To assist in understanding processes controlling migration in groundwater, and to illustrate the wide variation in potential impacts that might occur following a penetrator strike, a number of scenarios have been constructed for assessment using the ConSim groundwater contamination risk assessment model[1].

[1]Produced on behalf of the UK Environment Agency by Golders Associates and used in Environment Agency report (1999) *Contamination impact on groundwater – simulation by Monte Carlo method (ConSim)*. EA: Bristol.

Figure 9. Schematic diagram showing transport pathways associated with the contamination of groundwater supply from DU dusts or penetrators (not to scale). Note the use of the terms soil, unsaturated zone and aquifer that are use in the ConSim model. In the scenarios described in Annexe F the distance between the site of DU contamination and the water supply borehole is 20 m.



A wide range of alternative models could have been used of varying complexity. ConSim was chosen because its probabilistic approach enabled some of the wide variation in sorption properties to be incorporated into the model.

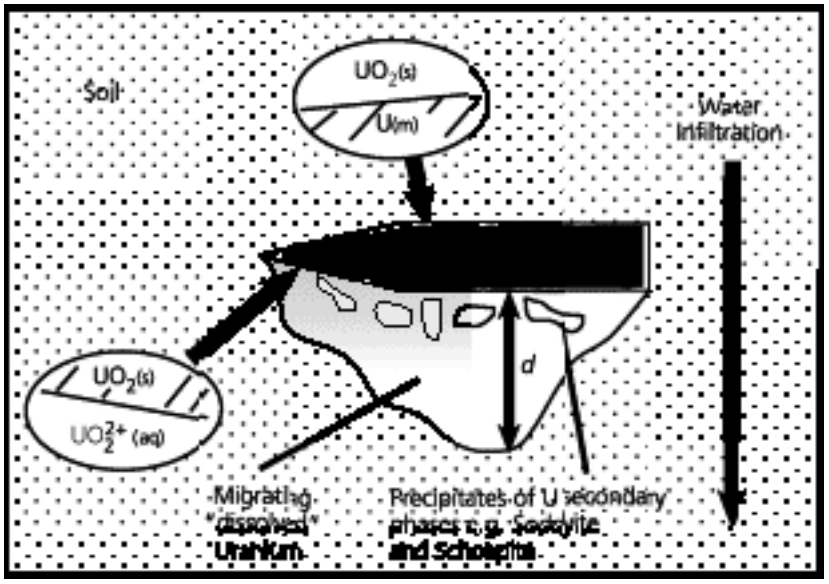
Two scenarios were selected as being representative of 'best-case' and 'worst-case' situations as defined below. Where appropriate (eg distances between strike sites and water supply wells), reference was made to conditions at strike sites reported in UNEP (2001) to link the developed scenarios to real-world situations.

- The best-case scenario (Uranium1) represents the best-case in relation to groundwater vulnerability; ie the uranium undergoes little chemical reaction from the U(IV) solid phases derived from the oxidation of uranium metal (U(0)), and in a low permeability matrix the reaction products are not transported very rapidly away from the penetrator site.
- The worst-case represents a much more permeable soil, which also allows oxygen ingression further into the aquifer, with inherently greater permeability allowing greater translocation of reaction products. The worst-case is used in Uranium2, and further modified by Uranium3 in which sorption in soil and the thickness of the unsaturated zone have been further reduced.

These scenarios and associated modelling are described in more detail in Annexe F. Results from modelling of scenarios Uranium1 and Uranium2 using ConSim are illustrated in Figure 11(a) and (b).

The most likely transit time for migration to reach a drinking water well sited 20 m from a DU strike site is around 30 million years (range one million to 100 million years) for the best-case scenario (Uranium1) and about 110 years (range 25 to 350 years) for the worst-case scenario (Uranium2). This difference is due to a combination of physical and chemical factors that inhibit the transport of uranium from the site of penetration. Both scenarios use similar distances from the penetrator strike to the water abstraction point (eg spring, water well or borehole). Further variability will be introduced if relatively simple site-specific information is included. For example, distances from strike sites to water supply wells are highly variable (less than five meters to greater than 100 m, UNEP, 2001), as are depths to groundwater, and as discussed in earlier sections of this appendix, considerable uncertainties exist in respect of the depth to which penetrators may have become buried in the subsoil. For example, the presence of deeply buried penetrators may reduce the period required for contamination to reach the base of the unsaturated zone from around 60 years (scenario Uranium2) to less than two years.

Figure 10. Schematic diagram illustrating initial corrosion and migration processes close to a corroding penetrator. Variation of the dimension, d, with time is dependent on local geochemical and hydrogeological conditions within a particular soil profile (s = solid, m = metal, aq = aqueous).



In addition to complicating the modelling of potential transit time, data uncertainty and scarcity also preclude the accurate prediction of likely concentrations of DU at the point of use (eg a water supply well). From scenarios modelled in Annexe F it is apparent that under worst-case conditions concentrations of DU at the point of use may exceed current recommended drinking water guidelines by at least one and potentially two orders of magnitude (scenarios Uranium2 and Uranium3, Annexe F).

The wide range of data, and the associated uncertainties of using averaged data, underpins the need for site-specific risk assessment when determining the vulnerability of drinking water supplies to DU contamination.

Even under worst-case scenarios (maximum uranium mobility and deeply buried penetrators), modelling performed in Annexe F indicates that it is unlikely that contamination of water supplies would be detectable until at least ten years have elapsed. Even then significant contamination may not be detectable until decades have passed. For this reason it important that: (1) negative results from the monitoring of water supplies immediately post-conflict (ie an apparent absence of contamination) should not be interpreted as indicating that future contamination is unlikely; and (2) that future monitoring strategies should be designed to test drinking water supplies over timescales of decades.

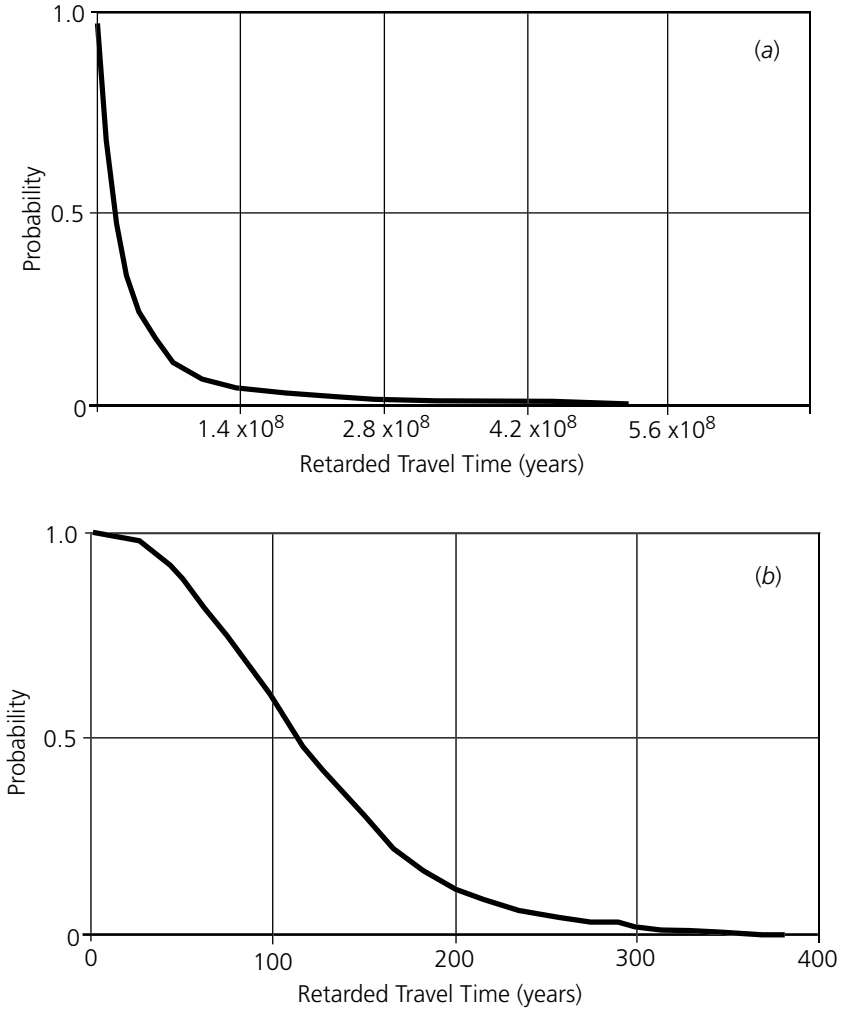
5.3 Case studies

5.3.1 Data and risk assessments based on proving grounds

The most extensively researched releases of DU into the environment have occurred in areas used by the military to test munitions (proving grounds). For example, an investigation at the US Army proving ground at Los Alamos suggested that up to 100 metric tonnes of DU may have been expended. It was estimated that a small canyon with an area of 3.1 square miles had a DU inventory in the region of 35 metric tonnes (Becker and Vanta 1995). Similar quantities of DU were also used at military proving grounds in Yuma, Aberdeen and Jefferson in the USA (Ebinger et al 1996; Ebinger and Oxenburg 1997). The use of DU munitions at the Kirkcudbright and Eskmeals sites has also been routinely monitored on behalf of the MOD since the early 1980s (MOD 1995), over which time it is estimated that 5000 test firings of various types of DU munitions have taken place (see also Section 2.3.2).

Although studies at such sites are useful for establishing the distribution of uranium immediately following dispersal, they provide little if any information about the longer term mobilisation and distribution of uranium because the studies have been in operation for less than 50 years (although detailed, reliable records of experiments and estimated releases of DU are probably only available for the latter half of this period). This timescale is relatively short, compared with those over which uranium dispersal and mixing occur (eg see Section 5.2 and Annexe F). The most practical way to undertake longer term studies is to investigate the dispersal of uranium at natural sites of uranium mineralisation. A wide range of such 'analogue' studies have been undertaken in support of the nuclear waste disposal industry, and have clearly demonstrated that oxides of uranium, including uraninite and pitchblende (UO_2), may be readily weathered by oxidation and complexation with inorganic and organic ligands and converted into more mobile, soluble, forms of uranium

Figure 11. Reverse cumulative probability from modelling of (a) Uranium1 and (b) Uranium2 scenarios using ConSim (1000 iterations).



that become incorporated into local surface waters, groundwaters, micro-organisms and plants (eg Basham et al 1989; Hooker et al 1989; Burns and Finch 1999). There is currently a lack of comparison between data produced from these studies and those derived from the DU alloys used in penetrators and associated particulates and aerosols.

5.3.1.1 Characterisation of contamination

Short-term leach testing of residues from munitions containing DU-Ti alloys at the Elgin test site (that had been used for test firing of DU munitions for over 20 years) indicated remobilisation of uranium from soils and to a more limited extent from drainage sediments over a timescale of 0 to 20 days (Becker and Vanta 1995). These authors hypothesised that the comparatively rapid leaching of uranium was due to the abundance of small particles released from the munitions during the combustion process (the majority of uranium particles being associated with the fine clay and silt fractions despite the sandy nature of the soil). Analysis of cores showed transport of DU to a depth with baseline uranium composition being reached at a depth of 100 cm.

Elless et al (1997) and Elless and Lee (1998) undertook a detailed characterisation of uranium-contaminated soils at various US DOE sites (eg the Fernald site in Ohio) that had been contaminated with uranium. Whilst uranium present at these sites was not associated with the use of DU-Ti alloys, the results clearly demonstrate the importance of considering the physiochemical form and bioavailability of soil-bound uranium when undertaking environmental and human health risk assessments. Uranium was found to be associated with the finer size fractions (silt and clays) of soil samples analysed in these studies. In addition, mineralogical analysis indicated that the predominant form of uranium contaminant in these soils was an autunite-like phase (eg hydrated calcium uranium(VI) phosphate). Major phase uranium minerals such as uraninite (UO_2, uranium(IV) oxide) and coffinite (uranium(IV) silicate, $USiO_4$) were also present. Whilst uraninite and coffinite are generally considered to be insoluble (less than 0.01 mg/litre), the dominant form, autunite, is only slightly soluble (0.1- 0.2 mg/litre) (Langmuir 1978).

During these studies (Elless et al (1997) and Elless and Lee (1998)), uranium solubility was determined before

and after remedial treatment in support of performing a health-based risk assessment. Solubility of uranium was determined in carbonate-rich soils associated with the contaminated sites, and in background soils, using 75- and 300-day extraction tests performed with rain and groundwater. The results indicated the importance of anionic uranium carbonate complexes in controlling mobility, and that the major control on uranium mobility was solubility control by primary mineralogical phases rather than sorption. The results also indicated that contamination of groundwater resources by DU derived from munitions was possible at the DOE Fernald site, and that this contamination was enhanced by the use of carbonate-based erosion control and road building materials. It should be noted that whereas a 75-day extraction test may be applicable to the leaching of uranium during infiltration of rainwater, it is inappropriate in assessing solubility within the human gastrointestinal tract, where residence times are in the order of hours (Ruby et al 1996). Similarly, the use of acid stomach simulants do not adequately account for dissolution of uranium in the neutral environment of the upper intestinal tract.

The longer term durability of relatively insoluble U(IV) oxides has been investigated during studies of the mobilisation of uranium dioxide stored in geological media with particular reference to the direct disposal of spent nuclear fuels (eg Cachoir et al 1996; Gallien et al 1996). Under oxidising conditions, a two-step process was defined in the alteration mechanism: (i) incorporation of oxygen and hydrogen correlated to a reduction in the volumetric uranium content (kinetic control); (ii) formation and dissolution of schoepite ($UO_3.2H_2O$) (thermodynamic control). Under reducing conditions, preliminary experimental results suggested an alternative mechanism. Gallien et al (1996) measured the concentration of uranium under reducing conditions to be as low as 10^{-11} molar. Other investigations, again undertaken during studies related to the disposal of nuclear waste, have investigated the occurrence and weatherability of uranium oxides under natural conditions (so-called 'natural analogue' studies). Such studies (eg Basham et al 1989; Hooker et al 1989) have shown that even reduced uranium oxides may over a period of tens, hundreds and thousands of years become mobilised into ecosystems and the local environment. These are timescales over which studies in the laboratory and at proving grounds are impractical or impossible.

5.3.1.2 Risk to ecosystems

Studies by Ebinger et al (1990; 1996) at the Aberdeen and Yuma Proving Grounds considered exposure to all components of the ecosystem and included both toxicological and radiological effects. Uranium was found in almost all samples and was present in most of the ecosystem compartments at Yuma (the semi-arid site) but not so many at Aberdeen. Measurable uranium concentrations were also found in aquatic endpoints (biota) at Yuma and in deer tissues at Aberdeen. Detection limits for ^{235}U precluded in most cases identification of this uranium as originating from DU munitions. However, uranium associated with some ecological endpoints could be clearly identified as being depleted in ^{235}U. Radiological effects were found to be insignificant at both sites but there was some tentative evidence of toxicological effects. Erosion at Yuma was demonstrated to be the primary mechanism of DU transport, with wind deposition being considered to be of secondary, and minor, importance. At the wetter Aberdeen site, the main migration pathway was the transport of suspended detritus in surface waters.

Concentrations of uranium in ecosystem components showed kidney content to be below threshold values in all species except for Kangaroo rats at Yuma (Ebinger et al 1996; pages 81 and 112), in which histopathology indicated possible damage to kidney tissue (Ebinger et al 1996; page 116). The consumption of dust, which had become adhered to foliage, was demonstrated to be the most important exposure pathway for animals living in these sites.

Model projections of exposure over the next 1000 years at these sites (Ebinger et al 1996; Ebinger and Oxenburg 1997) indicate a gradual decline in the importance of particulate exposure, together with a gradual increase in exposure to groundwater contamination over the next 100 years, before reaching a reasonably steady state condition between 100 and 1000 years (ie uranium particles become weathered, releasing dissolved uranium into the water table, or are physically removed from the area). Obviously such rates are extremely dependent on source term mineralogy, local soil type and hydrological conditions.

5.3.1.3 Risk to surface water and groundwater

Erikson et al (1990) reported on a number of early studies, several of which measured DU contamination in soils and groundwaters resulting from the impacts of DU penetrators at target ranges. For example, observations at the Los Alamos Scientific Laboratory (LASL) found uranium concentrations in standing water in detonation craters to range between 87 and 280 mg/litre, whilst concentrations in surface runoff water 100 m and 250 m from the site were 52 and 37 micrograms per litre (Hanson and Miera 1977). Reported concentrations in soil at the Aberdeen Proving Ground in Maryland (B-3 range and Fords Farm site) prior to 1980 were generally only marginally elevated over those expected for normal background concentrations, whilst concentrations in water were elevated by factors of up to 50-fold over expected background concentrations (Erikson et al 1990). In both cases contamination at the Aberdeen site was significantly less than at the LASL site described previously.

Ebinger et al (1996) investigated DU transport at the Aberdeen and Yuma Proving Grounds. Because the Yuma site is a desert environment with a deep water table, specific emphasis was placed on the potential migration of DU deposited on soils and eroded into desert washes and surface drainage. At the Aberdeen site a relatively shallow water table focused attention on migration through the soil and into groundwater. Previous studies at each site had indicated that: (1) DU migrates into the soil at the Aberdeen site because local rainfall is sufficient to promote transport; (2) DU migrates by way of soil erosion at Yuma; (3) no DU had been detected in groundwater at either site (note that the aquifer at Yuma is deep and hence migration over timescales involved would be minimal); and (4) sediments at Aberdeen showed some DU contamination whilst at Yuma DU contamination was detected in wash sediments.

Results of studies by Ebinger et al (1996) confirmed previous studies and emphasised the site-specific nature of the potential for groundwater and surface water contamination. For example, physical and chemical conditions at the Aberdeen site (low soil permeability, low Eh, high microbial activity) inhibited the corrosion of metallic DU-Ti alloys and subsequent migration into the sampling volume of monitoring wells (sited at up to four meters below ground level). At Yuma, despite soil conditions favouring the corrosion and transport of DU into groundwaters, the very low annual rainfall inhibited the transport of DU through the soil column. Thus in neither case was contamination of groundwater measured. Erosion was, however, demonstrated to transport DU at Yuma whilst at Aberdeen uranium in surface waters and associated sediments were shown to be contaminated with ^{235}U. In surface waters detritus contained the highest concentration of uranium, which in some cases could be identified as being depleted in ^{235}U.

At Aberdeen Proving Ground, modelling of uranium transport by Ebinger et al (1996) predicted the greatest concentrations of uranium from DU in surface waters and groundwaters to occur in 500 to 1000 years time.

5.3.1.4 Risks to human health

Risk calculations (based on both toxicological and radiological effects) and biokinetic modelling based on solubility measurements of uranium in contaminated soils at various US DOE sites contaminated with uranium rather than DU-Ti alloys (Elless et al 1997; Elless and Lee 1998) indicated that the risks were greatest from the soil ingestion pathway and the direct consumption of infiltrating groundwater. The lowest risks were attributed to the inhalation of soil-derived dusts. From the perspective of kidney toxicity, the greatest source of risk in studies and assessments at US proving grounds by Ebinger et al (1990; 1996) was derived from exposure due to the direct ingestion of infiltrating contaminated groundwater. In all cases, the calculated level of risk was extremely sensitive to the solubility of uranium and it was recommend by the authors that this parameter must not be overlooked when assessing potential risks associated with exposure to uranium from the environment.

Modelling of various exposure scenarios has been undertaken as part of environmental monitoring and decommissioning programmes carried out at US Army proving grounds that have become contaminated with DU. The Jefferson Proving Ground (JPG) decommissioning programme modelled exposure scenarios, which have been documented in several published reports, (Ebinger and Hansen 1994; AEPI 1995; Ebinger and Oxenburg 1997; Ebinger 1998; Oxenberg et al 1999). These studies only form examples of the results that may be obtained during case studies and should not be extrapolated to other sites, such as Serbia and the Middle East, without careful consideration and explicit justification. Three exposure scenarios were generally modelled in these studies to consider suitable uses for the site following decommissioning:

(i) An occasional user of the site visiting for four to six weeks of the year to hunt. The user would bring all food and water onto the site. The hunter would consume game animals.
(ii) A subsistence farmer consuming vegetables, dairy products and meat from crops and livestock produced on the site. Drinking water would be obtained from uncontaminated off-site sources. A fraction of the drinking water for livestock would be from contaminated groundwater, but the remainder would be from uncontaminated surface water.
(iii) As for scenario ii) except that all drinking water would be obtained from contaminated groundwater.

The modelling exercise concluded that no risk to humans occurred from occasional use of the site; the largest exposure to DU in this scenario was from exposure to contaminated dust (Ebinger et al 1996).

The farming scenarios showed some risk of exposure due to inhalation of contaminated dust, but by far the largest exposure resulted from the use of contaminated groundwater as drinking water, either by livestock or by humans. The overall conclusions of the modelling exercises were that subsistence farming presented a greater risk of DU exposure than did occasional use. However, in this particular study farming scenarios were not pursued in greater detail because farming and permanent occupation were considered to be inappropriate end uses due to the presence of unexploded ordnance on both proving grounds. Whilst such an assumption may be made in the case of proving grounds, similar assumptions cannot be made in areas of conflict where landmines and unexploded ordnance

Table 13. Investigation and action levels for DU in soil as used by the MOD for screening purposes (Gooding 1998)[1]

Limit	Bq/kg	mg/kg (assuming natural uranium)
First action level	300	11.9
Action level	1100	43.5
Radioactive Substances Act 1993[2]	11,100	439

[1]These are currently based on a small fraction of accepted GDLs for mixed soils (see Section 5.2) and therefore do not take into account the chemical toxicity of uranium.
[2]Level at which regulatory control is required for natural uranium.

have not prevented the areas being repopulated and farming activities being resumed (for example UNEP (2001)).

5.3.1.5 UK Proving Grounds
To date the study of the use and potential effects of DU at UK proving grounds has focussed on a strategy partly developed through a series of environmental reviews commissioned by the MOD during the mid-1990s (MOD 1995) and as a result of existing practices. The strategy has two main areas: (1) a well-defined temporal monitoring exercise to highlight any systematic increase in uranium content above a defined 'natural' background level; and (2) a limit-based (see Table 13) approach using established guidelines. Both of these areas have been supplemented by the use of measured uranium isotope ratios to identify the presence of DU.

During the 1990s, from which most of the monitoring has been reported, the first action level has rarely been exceeded at any site. This is very different from the situation at sites in the USA where concentrations in soils have been significantly elevated over natural background levels. It is, however, consistent with projectiles being fired out to sea in the case of Kirkcudbright rather than impacting with the terrestrial environment as is the case in the USA. An airborne gamma spectrometric survey commissioned by the MOD in 1995 showed no sign of an excess uranium burden at Kirkcudbright, although such surveys would not have been able to measure any uranium that had migrated to a depth in excess of 30 cm and hence would not have picked up any historical pre-1990 contamination.

Unlike studies in the USA virtually all of the focus at Eskmeals and Kirkcudbright has been on the potential radiological impact of the use of DU on humans and their associated food chain. Reference is made to the potential ecological effects in MOD (1995), but only limited studies appear to have been undertaken or reported to date (for example, on the faeces of various animals, including deer, hare, sheep and cattle). Studies of body burdens of small mammals and any potential detrimental effects, for example on kidney function, have not been undertaken, presumably because of the negative impact of such studies on the indigenous wildlife (as discussed in MOD (1995)).

Studies of the impact of fired DU rounds on the marine environment at Kirkcudbright have been limited by difficulties in identifying penetrators once they have become embedded in the soft marine sediments characteristic of the Solway Firth. Unsurprisingly given the relatively high abundance of uranium in uncontaminated sea water, and the potential for volumetric dilution, monitoring of sea water off the Kirkcudbright coast has not shown any increase in uranium concentrations over the past ten years of monitoring. Concentrations of uranium in marine sediments and biota again showed no enhancement of uranium levels from the uptake of dissolved or dispersed DU penetrators (shellfish, seaweed and bottom sediments; MoD 1995). Modelling of the transfer of DU through the marine environment using the best available data was undertaken on behalf of the MOD and suggests that exposures should be minimal (MOD 1995).

Despite numerous statements that the chemical toxicity of uranium is about the same as lead, no estimation or discussion of the likely relevance of the action levels outlined in Table 13 on chemical toxicity are made in MOD assessments of Kirkcudbright or Eskmeals, although with reference to Table 12 it is likely that the action levels outlined in Table 13 are protective to all apart from infants.

5.3.2 Data and risk assessments in areas of military conflict
A number of authors have used various theoretical scenarios to assess the likely human hazards posed by the use of DU munitions in conflict (eg Fetter and von Hippel 1999; UNEP 1999; Liolios 2000; SSI 2000). Results of these studies indicate that people at most risk of exposure to DU munitions are the occupants of vehicles attacked and penetrated by DU munitions. Members of the general population including those downwind of battlefields were not considered by these authors to be at risk of significant exposure, provided that vehicles struck by DU munitions were made inaccessible to curious civilians (or soldiers).

These studies lack validation and rely on relatively simplistic scenarios, complex modelling or low resolution broad-scale modelling due to lack of adequate data. Later studies such as those undertaken by CHPPM (2000) use more recent data, realistic scenarios and probabilistic models to describe uncertainty. As is the case with all such scenarios, they are subject to inaccuracies when considering site-specific issues that may enhance the potential exposure to DU (ie the heavy use of DU munitions in close proximity to important localised water resources or areas of market gardens).

5.3.2.1 The Gulf conflict

Few independent studies of the environmental impact and distribution of DU have been reported following the Gulf conflict. This is perhaps surprising given that much of the use of DU munitions in this particular conflict occurred in Saudi Arabia and Kuwait. Bou-Rabee (1995) measured uranium concentrations and isotopic ratios in eight air samples collected following the Gulf War (sampled in 1993–1994). The observed concentrations varied between 0.22 and 0.42 ng/m^3 with $^{235}U/^{238}U$ ratios ranging between 0.005 and 0.007. These concentrations lie within the expected background range, although the lower isotopic ratio (0.005) may be indicative of the presence of some DU.

Other data described in CHPPM (2000), AEPI (1995) and other publications associated with the Gulf War confirm on a site-specific basis the presence of various quantities of DU dust, penetrator fragments and intact penetrators associated with tank battles and the targets of air attacks in the days and months following the cessation of the conflict. The longer term mobilisation and migration of such source materials have not yet been systematically studied.

5.3.2.2 The Balkans conflict

Data are now being collated and reported from the Balkans conflict (eg UNEP (2001), MOD (2001), Sansone et al (2001) and a variety of other so-far unpublished studies by C Busby and Serbian investigators), and this is providing for the first time detailed site-specific data relating to the dispersion of DU from an actual conflict. However, the conflicts in the Balkans only involved the use of small calibre DU munitions used by the A10 attack aircraft and it is therefore impossible to use this particular conflict to assess potential impacts, or to support environmental transport models, associated with military campaigns (such as the Gulf War) in which larger calibre anti-tank munitions are also used or where much larger amounts of DU munitions are used.

The most marked observation from reports published to date is the very low proportion of penetrators apparently recovered (around 10 to 20%). This is consistent with most munitions becoming buried in the ground rather than hitting hardened targets and producing particulate oxidation products, and the exclusive use of A10 aircraft (30 mm DU munitions) to strafe military targets. All studies agree that local contamination with DU can be measured up to ten meters from a penetrator strike. However, elevated levels of uranium (ie above those of average soils) were generally restricted to less than one meter, and more typically less than 0.2 m, from the actual strike site. Given the variability of the approximately 250 potential impacts from a single multiple pass strafing attack, covering an area of say 200 m by 100 m, a high degree of variation in the energy dissipated and the production of DU-rich oxides would be expected. Absolute uranium concentrations at impact sites varied from a few mg/kg to in excess of 15 g/kg, a level at which significant local impacts might be observed in microbiota, plants and animals (see earlier). These areas of local contamination have been highlighted as potentially leading to elevated human (or animal) exposure via ingestion or local inhalation, as might occur if an infant was to be set down in the immediate vicinity of such a strike. These situations probably represent the only case where exposure is likely to exceed that estimated during a military conflict.

Depth profiles of soils from around penetrator impact sites indicated contamination of the soil to a depth of 20 cm. However, soil pore waters were not analysed to indicate concentrations of mobile uranium in infiltrating waters. Investigation of contamination from more deeply buried penetrators was not possible as these could not be located. Surprisingly no contamination of houses, vehicles or objects was noted in the UNEP mission, although the UK MOD noted some DU contamination of derelict buildings (MOD 2001).

To date, studies undertaken by UNEP in Kosovo (UNEP 2001) have not determined analytically the presence of DU contamination in either surface water or groundwater resources in the immediate vicinity of strike sites. However, studies were not undertaken to determine the presence of particulate or absorbed DU in river or lake sediments. Concentrations of uranium of natural origin measured in 18 water samples from Kosovo by UNEP did not exceed the WHO limit of two micrograms per litre and suggest that this value may be useful as a screening level for water supplies. Similarly, DU was not analytically determined in milk samples taken from cows grazing areas around strike sites.

During their investigations UNEP collected and analysed samples of grass, roots, tree bark, lichen and moss for DU. Results from these investigations were considered difficult to interpret because of the potential for contamination by entrained soil. Despite this, further investigations are being undertaken to investigate the presence of DU in lichen and its use as a biomarker for airborne DU contamination.

The UK MOD has now undertaken two field sampling exercises to Kosovo and data from these missions should soon be reported. Information released to date indicates the presence of particulate DU contamination where penetrators have impacted on concrete structures (Milodowski 2001) and that DU penetrators which impacted with concrete appear to have suffered minimal thermal oxidation (MOD 2001). It is interesting that such particulates can still be identified as superficial dusts after almost two years have elapsed since the penetrator impacts.

Preliminary data presented to the Working Group by Professor N Priest of Middlesex University (Priest and Thirlwall, personal communication) indicate that the presence of DU, presumably from military sources, is detectable by ICP-MS, at low levels, in members of the public selected as having potentially been exposed to DU from the conflicts in the Balkans. Further research is currently being undertaken to confirm or refute these important data, which suggest the existence of a pathway by which population exposure may, and possibly continues to, have occurred.

Kerekes et al (2001) undertook studies on the uranium content and uranium isotope ratios of airborne dusts from Kosovo in the atmosphere over Hungary. Whilst no characteristic signature of DU could be detected by alpha spectrometry, elevated levels of uranium with a natural isotopic signature were observed during the conflict, and these were attributed to well-dispersed dusts (2.5 microns in size) emitted into the atmosphere during bombing (a conclusion supported by the geographical and temporal distribution of measured concentrations). This study emphasises the potential for long-range transport should a large proportion of DU be converted to dust as a result of high energy hard target impacts occurring during military conflict.

6.0 Conclusions and knowledge gaps

DU is a radioactive material as defined in the UK by the Radioactive Substances Act 1993 and is classified as a List 2 substance by the EC Groundwater Directive due to its chemical toxicity. International limits covering human exposure to uranium in the environment have been defined by WHO from the perspective of chemical toxicity and WHO/IAEA with respect to its potential radiological effects. On the basis of available information it is likely that DU or uranium would be classified as a harmful substance under the Environment Protection Act 1990. Monitoring of the environmental impact of the release of large amounts of DU in military conflicts is therefore essential.

Immediately after its use on the battlefield, the main exposure of humans to DU is by inhalation and ingestion of the particles released from DU penetrators during impacts (or from shrapnel). However, people returning to, or continuing to live in, the battlefield will be exposed to DU from inhalation of DU particulates resuspended from contaminated soil and dust, and possibly over a larger timescale from contamination of water and food supplies by the uranium solubilised from DU particles, and from buried penetrators. Exposure from inhalation of particulates will reduce as DU is removed from the surface environment and, in the longer term, the environmental exposure pathways for DU become similar to the natural exposure routes where intakes from the ingestion of food, water or deliberate soil ingestion often dominate.

The chemical and mineralogical forms of DU released into the natural environment are difficult to characterise for every potential scenario, although the main endpoints are dusts of mixed DU oxides and metallic DU. In military uses, the chemical form and amounts of DU released into the environment are heavily dependent upon the nature of the penetrator impact (ie the type and composition of the penetrator, the energy of impact and the composition of the impacted material) and any subsequent changes due to the DU coming into contact with soil or water.
The nature and quantity of released DU have been reasonably well characterised during testing and on firing ranges. However, there are insufficient data to compare the composition and form of DU released under these controlled conditions with battlefield conditions. Since the first authenticated use of DU munitions was in the Persian Gulf War during 1991, there are very few data over environmentally significant timescales. For example, it is time periods greater than ten years, and more probably greater than 50 years, over which DU is likely to move significantly within the environment, leading to mixing with surface soils and groundwaters.

For the purposes of this appendix, the composition of DU released on the battlefield was characterised by considering two groups: uranium-rich particles (dusts) generated during impacts and subsequent fires, and residual metallic fragments and nearly intact penetrators.

The corrosion/dissolution rates of DU particles are relatively poorly studied compared with their dissolution in biological fluids. The relative importance of DU introduced as dust depends on the depth at which the material is introduced and then how much it is moved into the upper soil layers as a result of agricultural practices. If DU was restricted to the upper one cm or less of soil, as might be expected from the deposition of DU dust onto uniform soils of a high clay content, then the resultant concentration, assuming even airborne dispersal, would be in excess of 170 mg per kg. The restriction of elevated concentrations to the top one cm of soil is likely to increase transfer to some surface

rooting plants, and intakes by inhalation of DU from resuspension of soil and from ingestion of soil by grazing animals or by children (geophagy). It is therefore also important to consider the rate at which such dusts are transported or mixed within the upper layers of the soil. Such studies that have been undertaken on proving grounds or sites of military conflict have generally lacked a sufficient degree of spatial resolution or focus in this respect.

The depth to which DU projectiles penetrate into soil depends on the mechanical and physical properties of the soil and soil horizons. However, information on the relationship between penetration depth and soil characteristics has not yet been reported in the open literature. In Kosovo it has been considered that small calibre penetrators impacting into soft soil may penetrate into the ground to a depth of up to seven metres with minimal production of DU dusts (UNEP 2001). In some cases in the Gulf War large calibre penetrators fired from tanks were reported as going through their target without oxidising or producing substantial quantities of dust, resulting in relatively large pieces of metallic DU entering the environment. These uncertainties, coupled with difficulties in identifying DU penetrators that have missed their target and become embedded in the soil, represent a significant knowledge gap, particularly where targets have been strafed and the proportion of penetrators hitting a hard target is low.

After their deposition in the soil, the movement in the environment of uranium from DU dusts or intact fragments depends on the rate of corrosion and the rate of dissolution of the corrosion products. The corrosion and dissolution rates of DU dusts depend upon their chemical composition and size distribution. Uranium oxides constitute the main component of dusts produced from DU during impacts or fires, although such dusts may also contain a mixture of major or trace impurities such as iron, silicon and titanium. These impurities are not present in uranium dusts in the nuclear industry, so studies of the corrosion and dissolution of dusts from the nuclear industry may not necessarily be relevant to DU dusts.

DU in penetrators is alloyed with a small amount of titanium, which makes the corrosion properties significantly different from those of pure uranium metal. Alloying with titanium reduces corrosion and oxidation, retarding the release of soluble DU into the environment.

Much of our knowledge of the environmental behaviour of DU comes from studies at sites where DU munitions were tested. Based on measured corrosion rates, penetrators will only remain as metallic DU for between five and ten years. Reaction products from the corrosion of DU may be transported as a solid phase by physical processes such as resuspension or may be dissolved in soil water which may, depending upon local hydrological and environmental conditions, become transported into plants, surface waters or groundwaters. During the latter process, migration of dissolved DU is controlled by its solubility under local chemical conditions within the soil water and its sorption onto the immobile soil matrix (both of which may vary significantly over a scale of centimetres). Hence, corrosion rates, the solubility of the corrosion products and the degree of movement of DU in the environment will vary between locations and environments.

The behaviour of uranium is strongly affected by many environmental variables, such as soil composition and chemistry, the level of the water table, the amount of resuspension into the air, climate and agricultural practices. The large range in the possible values of these variables, together with the high degree of heterogeneity and uncertainty associated with the environmental distribution of DU from a military conflict, severely limit the applicability of generic models and site-specific models developed from existing data.

Most studies undertaken on proving grounds or in post-conflict situations suggest that atmospheric transport of DU occurs over relatively short distances (tens of metres) following the impact of armour-piercing DU projectiles. Longer range transport of airborne particulates (tens of kilometres) containing uranium with a natural isotopic signature have, however, been observed in at least one study of airborne uranium concentrations associated with the Kosovo conflict and in a number of studies in which uranium has been introduced into the atmosphere by nuclear fuel processing or coal combustion. Removal of DU particulates from the near-surface environment (where they may be resuspended) is likely to be relatively rapid, given the apparent corrosion rates. However, data collected in post-conflict assessments and proving ground studies suggest that particulate material may remain on or near the surface, even after two years have elapsed.

When introduced into the environment, DU is present in significantly different chemical and mineralogical forms to those encountered in natural systems in which much of the easily leached or 'labile' natural uranium has already been removed. In addition to being more easily leached, uranium derived from the fragmentation or corrosion of DU munitions may be more bioavailable, and possibly more mobile in the environment, than the residual uranium naturally present in weathered soils. The mobility of uranium released by weathering of DU is dependent upon the affinity of the soil for uranium and the properties of the soil. Thus, where soil strongly binds uranium, its release into soil water, and movement into groundwater, should be minimal. Correspondingly, mobility is likely to be greater in soils that bind uranium

less strongly. In environments where uranium is mobile, both point sources of DU, such as an intact penetrator or fragment, and diffuse sources, such as DU deposited from aerosols, will gradually disperse throughout the soil. Although this reduces contamination from DU in soil, the enhanced mobility implies that the level of contamination in groundwater may be increased. Similarly, such dispersal of DU may significantly decrease the cost-effectiveness and the technical feasibility of clean-up as a larger quantity of contaminated material may require disposal or treatment.

The primary factors affecting the potential for DU contaminating surface and/or groundwater resources, assuming that the uranium is mobile, are the proximity of the contamination to the water source (in the case of surface water) and the water table. For example, groundwater resources associated with river gravels may be particularly vulnerable due to their proximity to the surface. In contrast, the vulnerability of a deeper, possibly confined**,** underground body of water will be inherently lower. Perhaps the worst-case scenario with respect to groundwater contamination is that of a DU round penetrating the soil and lodging in a shallow groundwater system (such as an alluvial aquifer). This scenario may directly release uranium into a local water supply, such as a well, as the soil will not be able to act as a 'filter' to prevent any of the uranium entering the aquifer. However, unless the penetrator is directly lodged in a well, even with rapid dissolution such contamination may not be expected to result in a measurable increase in uranium concentration at the point of use until five to ten years have passed, even assuming reasonably conservative hydrogeological parameters. The best-case scenario with respect to groundwater or surface water is that the penetrator directly enters a highly sorbing medium such as soil with a high organic carbon content, or that it impacts in a clay-rich environment which is effectively impermeable to water, thereby preventing water flow and the migration of dissolved or particulate DU.

Most plants take up their nutrients (and contaminants such as uranium) mainly via the roots from the soil solution, although absorption through leaves also occurs. The extent to which uranium or DU is bound to soil components, and the strength of that binding, affects the amount of soluble soil uranium available for uptake into plants. Therefore, the factors influencing uranium mobility in soil are also likely to exert a strong influence on the extent of plant contamination. The soluble forms of uranium seem to be readily absorbed by plants, however in many soils natural uranium has a low solubility, and can be unevenly distributed. In general, uranium concentrations in plants decline in the order: roots greater than shoots greater than fruits and seeds. However, atmospherically deposited particulates including resuspended soil may significantly increase the concentration of uranium on foliage and unwashed fruits and seeds. The potential for contamination of plants is likely to be very variable due to the presence of highly localised contamination hotspots in soils associated with individual penetrator sites.

Concentration ratios that describe the relative concentration of uranium in plants compared with that in soil have been determined for various sources of uranium (eg mine wastes, tailings and nuclear fuel processing wastes). However, detailed investigations have not yet been reported that study DU-Ti alloys and their corrosion products. Although there are extensive compilations of data, the suggested concentration ratios vary by four orders of magnitude for the same crop on different soils and with different sources of uranium. This wide variation severely inhibits the applicability of generic models that incorporate uranium uptake into plants, and highlights the need for further studies with well-defined source terms and soil compositions.

The extent of absorption via the inhalation pathway in animals depends on the size and chemical form of the inhaled uranium, which influence the degree to which uranium penetrates the lungs and the rate at which it is solubilised in the lung. Uptake of uranium from the gut to the blood is low and, as in humans, most ingested uranium is excreted in faeces. Recommended gut uptake factors for ruminants are around five times higher than for monogastrics (eg humans). Once taken up the biodistribution of uranium in animals broadly follows that observed in humans and, compared with other body tissues, high concentrations have been reported in kidney, liver and tracheobronchial lymph nodes.

The dominant exposure pathways for humans, animals and plants are dependent upon the nature of the contaminative event and the time elapsed between the release of DU into the environment and exposure. For example, during a conflict, exposure of humans and animals in the immediate vicinity of penetrator sites will be dominated by inhalation, whilst exposure for those living in the vicinity of a combat zone 50 years later may be dominated by ingestion, since the contamination has settled out from the air and uranium has been solubilised from DU particles and buried penetrators, and become increasingly evenly dispersed amongst soil, plants and drinking water.
Of the many potential intake pathways associated with ingestion, exposure to DU via drinking water, milk and soil were considered to be the most important pathways. This was particularly the case in young children and infants. Unsurprisingly, in cultures where the deliberate ingestion of soil is practised, soil ingestion represents a dominant pathway even when the low bioavailability of uranium in soil is taken into account. This is because concentrations of uranium in

contaminated soil may be ten thousand times greater than those in drinking water. Where exposures are limited to accidental or everyday exposures to soils and dusts (eg finger to mouth contact), these form a less important pathway.

Monitoring of DU in the natural environment may be readily achieved through the use of modern methods of chemical analysis such as ICP-MS, which offer suitably low detection limits, accuracy and precision; at the same time they may be used to measure the isotopic composition of uranium to identify uniquely the presence of DU. The use of numerical modelling to predict environmental risks to human health and/or ecosystems is reliant on the provision of reliable, often systematic, information whose accuracy or uncertainty is well characterised. Such data remain currently unavailable for situations in which DU has been used in military conflicts. Because of this, modelling of environmental effects has been restricted to the derivation of generalised derived limits for uranium for radiological and chemical toxicity, and the use of modelling to demonstrate the sensitivity of predictive models of groundwater contamination to highly specific site variables such as geology and soil type.

Derivation of generalised derived limits for uranium illustrate the potential utility of this approach for setting appropriate standards on which monitoring programmes may be designed. Calculated data emphasise the duality of radiological and chemical toxicity, and indicate that whilst limits derived on the basis of chemical toxicity are protective towards radiological effects, they do not necessarily produce unachievable limits provided that potential receptor age groups are clearly defined.

Modelling of the contamination of groundwater resources and wells by 'ConSim' using best-case and worst-case scenarios, based on data collected by UNEP in Kosovo, highlights the wide range of potential input parameters that need to be collected prior to obtaining an even partially reliable model, and the sensitivity of the model to relatively simply measured parameters such as depth to groundwater. The modelling of groundwater contamination also highlighted the need for continued long-term monitoring of groundwater supplies unless the amount of DU remaining in the ground following DU attack can be better quantified; it also indicated that even low levels of mobile DU contamination of soil could result in groundwater contaminated with uranium to levels in excess of current WHO guidelines.

The most extensively researched releases of DU into the environment have occurred at firing ranges, or proving grounds. Case studies at these sites have utilised many techniques, from relatively simple temporal and spatial environmental monitoring against given target or threshold levels (often related to radiological rather than chemical toxicity), to more complex studies involving the use of environmental transfer models and sampling of animals and plants to determine the presence of harm. At the Jefferson Proving Ground in the USA the results of modelling concluded that negligible risk to humans occurred from occasional use of the site, the largest exposure to DU being from contaminated dust. Farming scenarios showed some risk of exposure due to inhalation of contaminated dust, but by far the largest exposure resulted from the use of contaminated groundwater as drinking water, either by livestock or by humans. The overall conclusions of the modelling exercises were that subsistence farming presented a greater risk of DU exposure than did occasional use. Projections of exposure over the next 1000 years at these sites indicated a gradual decline of the importance of contaminated dust together with a gradual increase in groundwater contamination over the next 100 years, before reaching a steady concentration between 100 and 1000 years.

Such evaluations are extremely dependent on the exact mineralogy, local soil type and water conditions. Calculated levels of risk were extremely sensitive to the solubility of the uranium and it was recommended by the authors that this parameter must not be overlooked when assessing potential risks associated with exposure to uranium or DU from the environment. Studies performed at proving grounds in the USA have not indicated substantive levels of toxicity amongst components of natural ecosystems associated with these environments.

Studies of potential exposure at military proving or testing grounds provide valuable data, but the density and nature of DU munitions use are often very different from those during actual conflict (on the basis of levels reported to have occurred in the Gulf and Kosovo conflicts). Whilst the relative importance of routes of exposure will probably remain broadly similar, this difference makes extrapolation of potential exposures, and ultimately health effects, between proving grounds and an actual conflict difficult.

Whilst few independent studies of the environmental impact and distribution of DU have been reported following the Gulf conflict, a relatively large number have been undertaken since the Kosovo conflict. The most marked observation from the reports reviewed was the very low proportion of penetrators recovered (around 10 to 20%). This is consistent with such munitions becoming buried in the ground rather than hitting hardened targets and producing particulate oxidation products, and the exclusive use of A10 aircraft (30 mm DU munitions) to strafe military targets. All studies agree that local contamination with DU can be measured up to ten meters from a penetrator strike. However, elevated levels (ie above the levels of uranium in average soils) were generally restricted to less than one meter, and more typically less than 0.2 m, from the actual

strike site. Absolute uranium concentrations at impact sites varied from a few mg/kg of soil to in excess of 15 g/kg, a level at which significant local impacts might be observed in microbiota, plants and animals. These areas of local contamination have been highlighted as potentially leading to elevated human (or animal) exposure via ingestion or local inhalation, as might occur if an infant was to be set down in the immediate vicinity of such a strike. They also could also provide a hazard if food plants are grown at these sites. These situations probably represent the only case where exposure is likely to exceed those predicted during a military conflict.

To date no studies have observed the presence of DU contamination in drinking water (private wells in the vicinity of strike sites), milk or vegetables, although one preliminary study has reported the presence of DU in human urine in potentially exposed members of the local population (Balkans conflict). Whilst it is not surprising that contamination of drinking water, milk and/or vegetables remains undetected (as the timescale of migration and mixing of DU in the soil and thence into groundwater and crops is likely to be in the order of tens or hundreds of years), the observation of DU in human urine, if positively confirmed, suggests that initial exposures to those living in the vicinity of an attack may have occurred through a more direct route such as the inhalation of particulates containing DU.

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